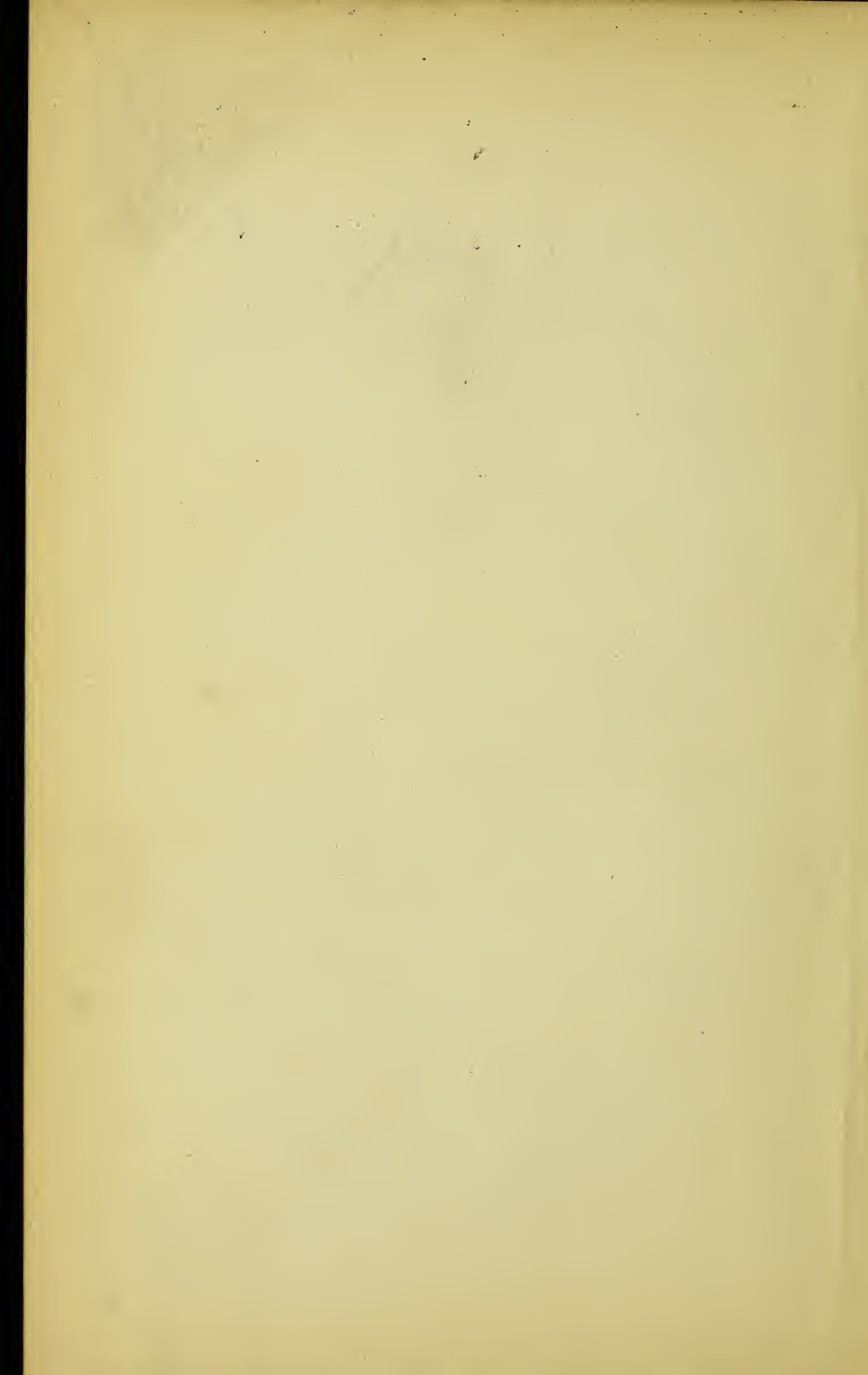




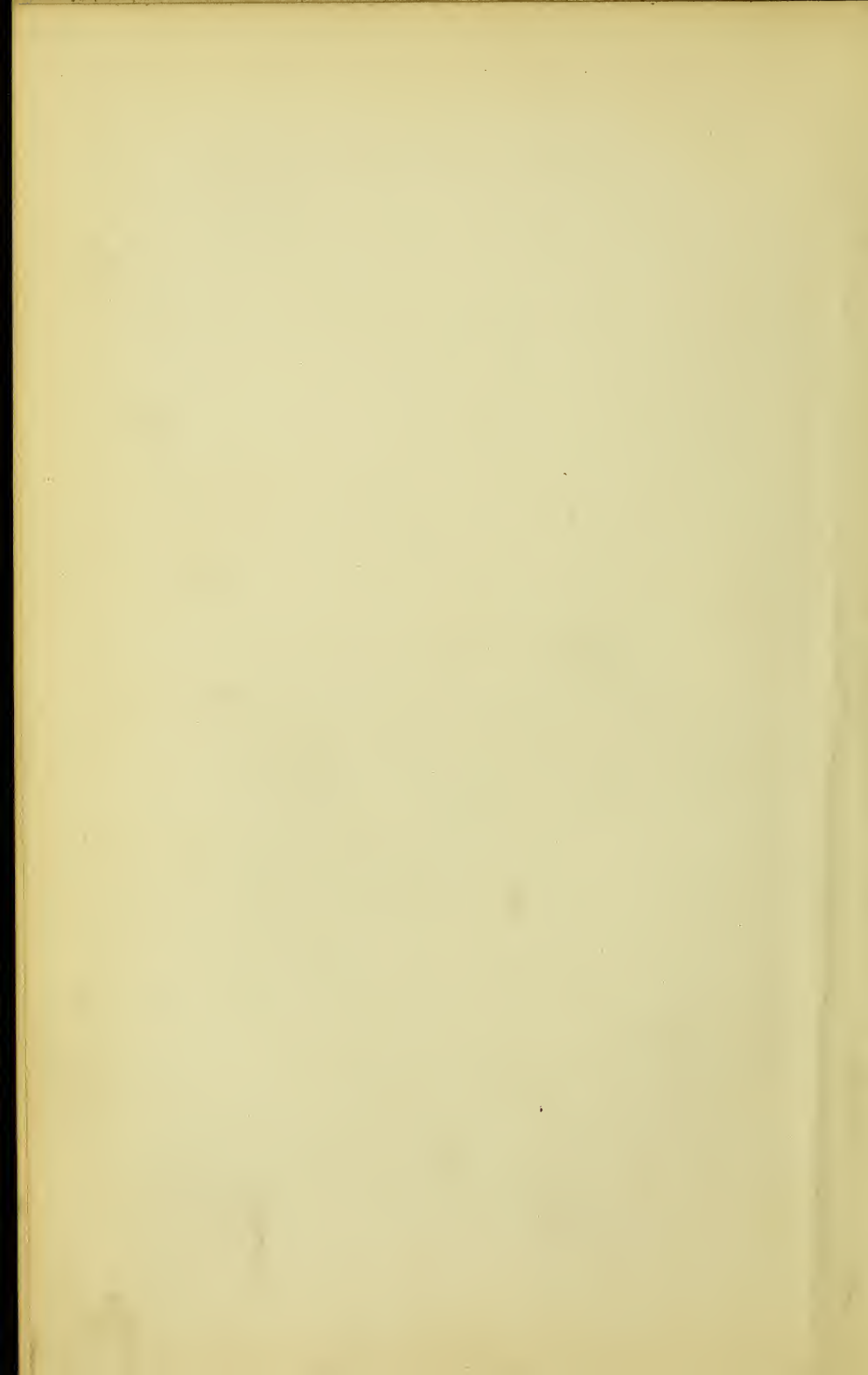
22101651770





W. J. Rees

A MANUAL
OF
INFECTIOUS DISEASES



79936

A MANUAL
OF
INFECTIOUS DISEASES

BY
E. W. GOODALL, M.D. LOND.

MEDICAL SUPERINTENDENT OF THE EASTERN HOSPITAL OF THE METROPOLITAN
ASYLUMS BOARD; FORMERLY MEDICAL REGISTRAR TO
GUY'S HOSPITAL

AND
J. W. WASHBOURN, M.D. LOND.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; PHYSICIAN TO THE LONDON
FEVER HOSPITAL; ASSISTANT PHYSICIAN TO GUY'S HOSPITAL,
AND LECTURER IN THE MEDICAL SCHOOL

LONDON
H. K. LEWIS, 136 GOWER STREET, W.C.
1896

8036

112 2667

LONDON

H. K. LEWIS, 136 GOWER STREET, W.C.

M17684

WELLCOME INSTITUTE LIBRARY	
Coll.	welMOmec
Call	
No.	Wc 100
	1896
	G 64m

P R E F A C E.

THIS book is designed chiefly for the use of students, for whom attendance in the wards of a fever hospital is now compulsory. But while it is intended to supplement bedside teaching, the authors hope that it will prove helpful to those who have little or no time to spare for special courses of instruction, such as are now held in most of the large fever hospitals in London and the provinces.

The book treats of the specific fevers common in this country, and includes, in addition, chapters upon Typhus Fever, Relapsing Fever, and Anthrax. Bearing in mind the history of Influenza in England, the reappearance of Typhus and Relapsing Fever is quite possible; and the latter is an excellent example of an epidemic infectious disease due to a micro-organism belonging to the class of Protozoa. Anthrax was chosen as a type of a bacterial disease derived from animals.

Short chapters have been devoted to the discussion of Fever, Contagion, and Disinfection; and, on account of the frequent mistakes made in diagnosis, we have thought it advisable to give a brief account of Sore Throat and of the Rashes simulating those of the Specific Fevers.

In accordance with the practical object of the book, we have paid considerable attention to diagnosis and

treatment, and have only dealt with the pathology so far as it throws light upon these questions.

The Appendix contains, in addition to other matters, a *résumé* of a Report of the Medical Officers of the Metropolitan Asylums Board Hospitals upon the Antitoxine Treatment of Diphtheria, which was published while this book was in the press.

The figures representing the distribution of rashes are purely diagrammatic. They are intended to demonstrate the extent of surface usually affected by the rash in the various diseases, and they do not represent the characters of the skin lesions.

We beg to express our thanks to Dr. H. E. Durham, the Gull Student in Pathology, for the majority of the photographs; and to Dr. E. A. Peters for the preparations of the Diphtheria Bacillus, the photographs of which were taken by Mr. Bousfield.

Most of the temperature charts are from cases that have been under the care of the authors; but for the chart of Typhus Fever we are indebted to Dr. Copeman, and for those of Small-pox to Dr. Ricketts, Medical Superintendent of the Hospital Ships at Long Reach. We have also to thank Dr. Ricketts for many valuable suggestions in the chapter on Small-pox.

To our friend Mr. George Rowell, F.R.C.S., Anæsthetist to Guy's Hospital, we owe special thanks. He helped us to revise both the manuscript and proof sheets, and has given us great assistance in passing the book through the press.

Dr. Potts, of the Eastern Hospital, has also assisted us in the matter of proof-reading.

E. W. G.

J. W. W.

CONTENTS.

CHAP.	PAGE
I. FEVER	I
II. CONTAGION AND INFECTION	15
III. DISINFECTION	41
IV. ON RASHES SIMULATING THOSE OF THE SPECIFIC FEVERS.	49
V. ON SORE THROAT	53
VI. SCARLET FEVER. SCARLATINA	57
VII. DIPHTHERIA	110
VIII. MEASLES. MORBILLI	165
IX. RUBEOLA. ROETHELN. GERMAN MEASLES	181
X. VARIOLA. SMALL-POX	188
XI. VACCINIA	218
XII. VARICELLA. CHICKEN-POX	230
XIII. WHOOPING-COUGH. PERTUSSIS	242
XIV. MUMPS. SPECIFIC PAROTITIS	249
XV. EPIDEMIC INFLUENZA	253
XVI. TYPHUS FEVER	265
XVII. RELAPSING FEVER. FAMINE FEVER	279
XVIII. ENTERIC FEVER. TYPHOID FEVER.	290
XIX. ERYSIPELAS	330
XX. ANTHRAX. MALIGNANT PUSTULE. WOOL-SORTER'S DISEASE. SPLENIC FEVER	340

LIST OF PLATES.

PLATE I.

SIX PHOTOGRAPHS OF THE DIPHTHERIA BACILLUS . . . *To face page* 138

PLATE II.

TWO PHOTOGRAPHS OF THE INFLUENZA BACILLUS . . . *To face page* 260

PLATE III.

THREE PHOTOGRAPHS OF THE TYPHOID BACILLUS, AND ONE OF THE
BACILLUS COLI *To face page* 318

PLATE IV.

TWO PHOTOGRAPHS OF THE STREPTOCOCCUS ERYSIPELATIS *To face page* 336

PLATE V.

FOUR PHOTOGRAPHS OF THE ANTHRAX BACILLUS . . . *To face page* 347

INFECTIOUS DISEASES.

CHAPTER I.

FEVER.

A WARM-BLOODED differs from a cold-blooded animal in the fact that its temperature remains at a fairly constant level under varying conditions of the external temperature. In man the normal temperature ranges between 97.8° and 98.8° Fahr., the highest point being reached towards six o'clock in the evening, and the lowest about two o'clock in the morning. In children the range is larger and the variation less constant.

We shall have to consider, firstly, the way in which the body temperature is maintained; secondly, the way in which it is regulated; and, lastly, the way in which it is altered during fever.

PRODUCTION AND LOSS OF HEAT.

The heat of the body is supplied by the oxidation which is constantly occurring in all the tissues during their destructive metabolism. All the active tissues are sources of heat to the body; but the muscles and such large glands as the liver contribute by far the largest share. During the contraction of the muscles a large amount of heat is produced, and if the muscles of an animal are tetanised by stimulating the spinal cord, the

temperature rapidly rises many degrees above normal. But apart from contraction the muscles are constantly producing heat; and when we consider their bulk in relation to the other tissues, there can be little doubt that they are the chief source of the heat of the body. Next to the muscles come the secretory glands; and it has been shown that the blood in the hepatic veins, as they leave the liver, is hotter than any other part of the body.

The amount of heat produced can be measured directly or indirectly. Indirectly, it is measured by estimating the carbonic acid given off, or more accurately the oxygen consumed; and from these data the amount of heat produced can be ascertained. The production of heat can be directly measured by a calorimeter, which consists essentially of a large metal chamber, in which the animal is placed. This chamber is surrounded by a narrow chamber containing air, and from the expansion of this air the amount of heat given off by the animal can be estimated. Provided the temperature of the animal remains constant during the experiment, the heat given off is equal to that produced, and thus the production of heat can be measured.

While heat is constantly being produced in the body it is also constantly being lost. The chief channels of loss are the skin and the lungs; for we may neglect such slight loss as occurs through the warming of the urine and fæces. From the skin heat is lost by conduction, radiation, and by the evaporation of the sweat; from the lungs heat is lost by the warming of the expired air, and by the evaporation of its moisture. It has been estimated that of the total heat lost more than three-quarters passes through the channels of the skin.

REGULATION OF TEMPERATURE.

The amount of heat lost through the skin depends, *cæteris paribus*, upon the condition of its blood-vessels and the quantity of sweat secreted. When the vessels are dilated warm blood from the deeply seated tissues is brought to the surface, and thus more heat is lost; and the same effect is produced by an abundant secretion of sweat, which leads to an increased evaporation and consequent abstraction of heat.

Both the dilatation of the blood-vessels and the secretion of sweat are directly under the influence of the central nervous system; and in this way the amount of heat lost through the skin can, within certain limits, be regulated.

The production of heat is also controlled by the central nervous system. Injuries to certain parts of the brain, such as the corpus striatum, cause a rise of temperature, which calorimetric observations show to be due to an increased production of heat. And when an animal is poisoned by curare, and the muscles, through paralysis of their nerves, are shut off from the nervous system, the temperature falls. This fall of temperature is shown to be due to a diminished production of heat by the fact that it is accompanied by a diminished elimination of carbonic acid.

In health the mechanism which presides over the production of heat and that which presides over its loss are so adjusted, that when one of these factors varies the other varies in the same degree. Thus the amount of heat lost in any given time is always equal to that produced; and, consequently, the temperature of the body remains constant. An example will illustrate the way in

which these two mechanisms are correlated. Muscular exercise, by increasing the metabolism, causes a large production of heat, and this would raise the temperature of the body were it not compensated by an increased loss of heat. Such a compensation occurs; for the blood-vessels of the skin become dilated, and sweat is poured out from the glands. Consequently, even with violent exercise, no appreciable change in the body temperature ensues.

SYMPTOMS OF FEVER.

In disease the normal temperature of the body is frequently altered. The most usual change is a rise of temperature, and when this exceeds 99° Fahr. the patient is said to be suffering from *pyrexia* or *fever*. It would perhaps be better to use the term *pyrexia* to connote simply an increased body temperature, and to apply the term *fever* to a group of symptoms of which the most obvious is *pyrexia*. Using the terms in the above sense, we will first discuss the clinical aspects of fever. If the temperature is observed throughout, several stages may be recognised; and information of diagnostic value can often be obtained by studying the way in which the temperature behaves during each stage.

The first or *initial stage* is that in which the temperature rises to its maximum. The duration of this stage differs according to the nature of the disease. In pneumonia it only lasts a few hours, the temperature rapidly reaching the maximum, while in enteric fever its duration is several days.

The second stage, the *acme* or *fastigium* as it is called, is that in which the temperature remains near its maximum

point. In some diseases, as in pneumonia or typhus fever, the fastigium is sharply defined from the initial and from the succeeding stage; but in others the different stages run into one another so imperceptibly that their exact separation is impossible. Sometimes during the fastigium the temperature remains at about the same level throughout; but usually marked morning remissions and evening exacerbations, exaggerating the normal variations, occur. Sometimes decided variations occur from day to day, and the pyrexia becomes of an irregular type.

The third stage is that of *defervescence*, during which the temperature falls to normal. It may be of short or long duration. In the former case the fever is said to end by *crisis*, and in the latter by *lysis*.

After defervescence the temperature often continues to fall, and remains subnormal for several days.

A comparison of the curves of temperature in different diseases affords interesting results; and it would appear that every specific fever yields a characteristic temperature curve, which, however, may be changed by a variety of circumstances. Comparing, for example, the curve of typhus (Chap. XVI.) with that of enteric fever (Chap. XVIII.), we see that in the former disease the temperature rises suddenly, keeps at its maximum for about a fortnight with but slight remission, and then falls quite suddenly to normal; while in the latter disease the temperature rises slowly, keeps at its maximum for about a fortnight with marked morning remissions and evening exacerbations, and then gradually falls again to normal. But in enteric fever the temperature curve is often extremely irregular, by no means conforming to the characteristic type above described, and this is doubtless due to the

frequency of secondary infection with other micro-organisms than that which causes enteric fever. It is, indeed, generally such secondary or associated infections which give rise to deviation from the normal types of temperature in the specific fevers.

Fever is attended with various other symptoms in addition to pyrexia. Some of these symptoms are due to the pyrexia, while others are concomitant symptoms due to the same causes as those which produce the pyrexia.

If the temperature rises suddenly the patient feels cold, and may be seized with a rigor, during which he shivers, and the skin becomes cold and blue from a contraction of the cutaneous vessels. The papillæ of the skin also become unduly prominent, producing what is called the *cutis anserina*. The sensation of coldness is due to the condition of the skin; for if the temperature be taken in the rectum during a rigor it will be found to be several degrees above the normal. After a time the cutaneous vessels dilate, the skin becomes warm, and a sensation of heat is experienced. Sometimes the skin remains quite dry, and sometimes there is profuse perspiration.

The pulse and the respirations are increased in proportion to the pyrexia. Roughly speaking, for every degree Fahrenheit, the pulse rate is increased by four beats above the normal of eighty.

The blood becomes less alkaline, and leucocytosis occurs. It has been shown that the injection of bacteria and their products into the circulation of animals produces a temporary diminution, followed by a more lasting increase of the leucocytes of the blood. The same effects are caused by the induction of local inflammatory changes

without the intervention of bacteria. In the human subject leucocytosis is observed in almost all infectious fevers. Enteric fever is an exception; for in this disease leucocytosis does not as a rule occur, and when found is probably due to septic complications. In some diseases, such as pneumonia, a marked leucocytosis is a favourable sign, while in others, such as diphtheria, the reverse appears to be the case.

Excessive thirst is a prominent symptom. The tongue is furred, the bowels constipated, and the appetite lost. The patient complains of headache, and pains in the back and limbs, is restless and sleepless; and there may be delirium, and even coma or convulsions.

The urine is scanty, of a dark colour and high specific gravity, and gives an abundant deposit of urates. A trace of albumen is often present when the temperature is high. Sometimes a diminished excretion of chlorides occurs.

If the fever continues for long, marked wasting of the tissues ensues, and degenerative changes of the nature of cloudy swelling occur in the various organs. To these changes is added an increased excretion of urea and carbonic acid.

THEORY OF FEVER.

The question arises as to how far the above-mentioned symptoms are directly due to the pyrexia. Are they caused simply by the increased temperature of the body? This question we may at once answer in the negative. A temperature which in one disease would be accompanied by pronounced symptoms of fever may be found in another disease without causing any marked

disturbance. In the so-called hysterical pyrexia the temperature may be many degrees above the normal without any of the other symptoms of fever. A patient suffering from phthisis may be quite comfortable with a temperature of 103° Fahr.; while a patient suffering from typhus fever with the same temperature will be lying prostrate in bed, with marked febrile symptoms.

We must thus look upon fever as a complex phenomenon attended by various symptoms, one of which is pyrexia. And in this connection it is interesting to note that one and the same pathogenic bacterium has been shown to produce several toxins, some of which cause pyrexia and others give rise to other symptoms. It is probable that any given bacterium generally produces these different toxins in the same proportion; and thus in any given specific fever the height of the pyrexia gives us a fairly good indication of the severity of the attack. A notable exception occurs in diphtheria; for in this disease a high temperature does not indicate a severe attack nor a low temperature a mild attack. But here we are probably dealing with a mixed infection.

Now although we have insisted upon the fact that the symptoms of fever are not simply caused by the temperature, yet it must be admitted that a very high temperature by itself will produce a train of symptoms similar to those we have described above. We have already pointed out that the rapidity of the pulse and respiration in fever is roughly proportional to the temperature, which would suggest a causal connection between them. And, indeed, the exposure of the isolated mammalian heart to various temperatures has been shown to change the rapidity of the beat in pro-

portion to the temperature. Again, in hyperpyrexia from various causes, when the temperature is lowered by the application of ice the other symptoms subside. So that it would appear that pyrexia has a certain influence in causing the other symptoms of fever; but this influence in most cases is only of subsidiary importance.

In discussing the regulation of the temperature of the body we have seen that a rise of temperature may be caused either by an increased production or a decreased loss of heat. We shall have to consider which is the principal cause of the pyrexia of fever. The experimental evidence is too scanty to allow us to draw definite conclusions; and we shall be compelled to resort to reasoning from analogy in framing our judgment.

During the initial stage of pyrexia the rise of temperature is, to a large extent, due to a diminished loss of heat caused by the contraction of the vessels of the skin. There is probably also an increased production of heat, which, in the case of a rigor, is assisted by the muscular contractions during the shivering stage.

But during the second stage, when the temperature has risen to the maximum, and is remaining at a high level, there is no longer a diminished loss of heat, but, on the contrary, an increase. For the vessels of the skin are dilated, the secretion of sweat is often increased, and, besides, on account of the high temperature, the body will lose more heat by conduction and radiation. Also the rapidity of respiration causes more heat to be lost through the lungs. With this increased loss of heat there must be an increased production, otherwise the temperature, instead of remaining high, would fall below the normal. Calorimetric observations, so far as they go,

support this view ; and so too does the fact that there is an excessive output of carbonic acid and an increased consumption of oxygen. The increased excretion of urea is due to the wasting of the tissues, and does not appear to bear a causal relation to the pyrexia. The mechanism presiding over the relation between the production and loss of heat remains intact during the fastigium, but is set at a higher level. It is, however, in an unstable condition, as is shown by the frequent remissions and exacerbations of the temperature.

During the stage of defervescence the heat production sinks to normal, and there is frequently also an increased loss of heat, due to profuse perspiration.

Though the course of events is usually such as we have described, yet there may be temporary forms of pyrexia, due entirely to a diminished loss of heat ; but when the pyrexia is long maintained there must be also an increased production of heat.

The term *hyperpyrexia* is used in a different sense by different authors. By some it is used to denote any temperature which in itself is dangerous to life ; others fix some arbitrary limit, such as 106° Fahr., and call any temperature above this point hyperpyrexia ; by others the term is used to denote a special condition, differing in essential points from ordinary pyrexia. This condition occurs in certain cases of rheumatic fever, and is characterised by the fact that the temperature steadily rises until it reaches such a height as, unless it be artificially reduced, leads to death. The mechanism presiding over the regulation of production and loss of heat is upset, and the rise of temperature appears to be due more to this upset than to the increased production of heat, which undoubtedly also occurs.

CAUSATION OF PYREXIA.

As to the exciting causes of pyrexia, there are several ways in which this condition can be experimentally produced. They are :—

1. Injuries to the central nervous system, especially to the corpora striata, the pons, and the upper part of the spinal cord.
2. Exposure of the body to a very high external temperature.
3. Inoculation with various bacteria.
4. The injection of various bacterial toxins, and of certain proteids, such as albumoses.

TREATMENT OF PYREXIA.

In considering the treatment of pyrexia we must first ask the question whether the pyrexia is in itself harmful. It is generally assumed that the pyrexia is the cause of the other symptoms of fever, and that any treatment which will lower the temperature must be beneficial. As we have already pointed out, this view is incorrect ; and, besides, we must admit that the systematic use of antipyretic treatment during the infective fevers has not led to any better results than the expectant treatment. On the other hand, it is quite possible that pyrexia may be a beneficial process, assisting the body in coping with the micro-organisms of disease ; for it is found that many bacteria are attenuated by exposure to high temperatures.

A very high temperature such as occurs in hyperpyrexia is however decidedly harmful, and vigorous treatment will often prevent a fatal termination. Sometimes, too, even with a moderate pyrexia, the artificial lowering of

the temperature is of benefit in allaying headache and discomfort, and in procuring sleep; but to adopt a systematic antipyretic treatment does not appear to be of any advantage, and it may even do harm.

Pyrexia may be reduced (i) by the administration of certain drugs; (ii) by the application of cool or cold water to the skin; (iii) by cooling the air surrounding the patient; (iv) by the application of ice to the skin.

(i) The action of antipyretic drugs is very uncertain, and varies much both in the various febrile diseases and in the same case at different times. The drugs to be recommended are sulphate of quinine (gr. i. to gr. x.), phenacetine (5 to 10 grains), antipyrin (5 to 20 grains), and antifebrin or acetanilide (2 to 5, or even 10 grains). Of the last three drugs the safest is antifebrin; the others are liable to produce collapse.

(ii) Cold water is applied to the surface of the body by sponging, by wrapping the patient in wet sheets (the wet-pack), or by immersing him in a bath.

To *sponge* a patient envelop him loosely in a blanket, and sponge thoroughly the skin of the trunk and limbs for a quarter of an hour with a sponge partly wrung out of tepid or cold water. The sponging may be repeated every three or four hours, or when the temperature reaches a certain height.

To *wet-pack* a patient he should be wrapped (trunk and limbs) in a sheet wrung out of tepid, cold, or even iced water. He is laid on a blanket and covered by a couple of blankets. At the end of twenty minutes or half an hour the sheet is removed, the skin quickly dried with a towel, and the patient placed between dry blankets. In certain cases the patient may with advantage be kept in the wet-pack for several hours or days. In these cases the

sheet should be wrung out of tepid water, and should be wrapped round the trunk and proximal portions of the extremities, the distal portions of which should be enveloped in cotton-wool. The sheet should not be changed unless it becomes soiled, and may be kept moist by having water squeezed upon it from a sponge.

In the *cold-bath* treatment the patient is wrapped in a blanket and immersed in a bath at 90° Fahr. Cold water or ice is then gradually added to the bath. The temperature of the patient should be taken by means of a thermometer in the rectum, and he should be taken out of the bath as soon as it has fallen to 101° Fahr. He should not be kept in the bath for longer than half an hour.

Dr. Barr, of Liverpool, recommends the *continuous bath* for certain cases of enteric fever. The patient is kept immersed in a specially constructed tank for several days, or even three or four weeks. The temperature of the water is from 90° to 98° Fahr.

(iii) The air surrounding the patient can be cooled by means of the *ice-cradle*. The patient lies with his trunk and limbs under a large cradle, such as is used for surgical cases. He is either naked or covered with a sheet of gauze. Inside the cradle are hung three or four zinc pails filled with ice. The cradle is covered with a blanket or counterpane, an opening being left at each end for the free exit and entrance of air. The patient may be kept in the cradle for many days.

(iv) *Ice* may be applied either in india-rubber bags or spread upon lint or a towel. The bags are placed either on the abdomen or the axillæ, groins, and head. But ice is best applied to the abdomen in the following way. Lay upon the abdomen a towel or large piece of lint. Roll the margin of the towel or lint round some cotton-wool to

catch the water that runs away from the ice as it melts. Break the ice into small pieces and spread it on the towel, which should be left exposed in order that the ice can be seen to melt. The ice is renewed as it melts. With the india-rubber bag the ice has often become warm water long before the nurse has discovered that it has melted. The patient's limbs should be wrapped in cotton-wool.

Cold may also be applied to the skin by means of coils of india-rubber or metal pipes, through which cold water is continuously running (Leiter's coils).

In all the above methods of treatment by the application of cold the patient should be very carefully watched, for collapse will sometimes result. It is often advisable to administer a stimulant during the treatment.

CHAPTER II.

CONTAGION AND INFECTION.

DISEASES which are transmissible from one individual to another are said to be *contagious* or *infectious*. Strictly speaking, the term infectious is applied to diseases transmissible through the air, while the term contagious is used of diseases which are spread by direct contact, or by infected articles (fomites). In this sense small-pox would be called an infectious, and anthrax a contagious disease; for the former is usually conveyed through the air and the latter by infected articles. But small-pox can infect by direct contact, as in the old preventive treatment of variolation, and anthrax may be conveyed by the air, as occurs in certain forms of wool-sorter's disease. The distinction between infection and contagion thus breaks down, and it is perhaps better to give it up altogether. Nevertheless, we must not lose sight of the fact that some diseases are generally conveyed through the air, and others by direct or indirect contact.

With regard to the *nature of the contagium*, there can be little doubt that the *materies morbi* is always a living micro-organism. In some diseases—as, for example, relapsing fever, diphtheria, and glanders—this has been definitely proved; but in others—for example, scarlet

fever and measles—we can only surmise that it is so by analogy.

As to the nature of the pathogenic micro-organisms, those with which we are best acquainted belong to the class fungi, and especially to the bacteria. But the organism peculiar to relapsing fever belongs to the class protozoa; so that possibly it will be found that the organisms of other infectious fevers belong to the same order. These micro-organisms occupy the lowest grade of life, at the point where the animal and vegetable kingdoms meet. A short description of them is necessary, and we will begin with the protozoa, although we can dismiss them in a few words.

Protozoa.—The protozoa are micro-organisms belonging to the lowest forms of the animal kingdom. They consist of amœboid masses of protoplasm, which multiply either by division or by the formation of spores. When spore formation occurs the amœboid mass often becomes surrounded by a distinct cell-wall previously to breaking up into spores.

One of the best examples of pathogenic protozoa is the malaria parasite.

Fungi.—The fungi are the lowest forms of vegetable life, and are divided into several classes, of which the bacteria are the most important from a pathological point of view.

CHARACTERS OF BACTERIA.

The bacteria are unicellular organisms with a cell-wall and protoplasmic contents. *Reproduction* takes place either by *division* or by the formation of *spores*. In the former method of multiplication one or more partitions form in the mother cell, and divide it up into two or more

daughter cells. These remain at first united together, but ultimately they separate, and thus form new individuals.

Spores are round or oval, highly refractile bodies, which are formed in the interior of the bacterium, and at the expense of its protoplasm. After a time the cell-wall and the remains of the protoplasm undergo a process of solution, and the spore is set free. Under favourable circumstances the spore germinates, and produces a new individual, having all the characters of the original cell from which it has sprung. All bacteria multiply by division: spore-formation is only observed in certain species under certain conditions of growth. Spores are characterised by possessing a high resistance to the action of various destructive agents, such as heat, drying, and chemicals. They are thus well adapted for preserving the existence of the bacteria when placed under unfavourable external conditions.

Some bacteria, although not forming spores, yet produce under certain circumstances highly resistant individuals, which perform the same function of preserving the life of the species.

For the sake of convenience the bacteria can be classified, according to their shape, into *bacilli*, *vibrios*, and *cocci*.*

The *bacilli* are rod-shaped or filamentous bodies, multiplying by division in a direction transverse to their long axis. After division they frequently remain united together, so as to form long jointed threads. Some species form spores. Some bacilli are motile, others are not.

Vibrios are short twisted rods like one turn of a cork-

* Bacterium, βακτήριον = βάκτρον a staff or rod. Bacillus means a small rod. Vibrio is apparently derived from vibrare, to vibrate. Coccus = κόκκος, a berry.

screw, and are usually motile. After division, the new individuals may remain united together spiralwise, like several turns of a corkscrew. They are then often called *spirilla*. The term "comma-bacillus" is sometimes given to the separate individuals.

The *cocci* are round or oval non-motile bodies. They are sub-divided into the following forms according to the mode of multiplication. When division occurs in only one direction, and the resulting cocci are associated in chains, we call them *streptococci* (στρεπτός, twisted; a chain, necklace); if in small irregular masses, *staphylococci* (σταφυλή, a bunch of grapes); and if in pairs, *diplococci*. When division occurs in two directions at right angles, and the cocci remain associated in fours, they are called *tetrads*; while if division takes place in three planes, and the cocci remain together in packets, they are called *sarcinæ* (*sarcina*, a bundle). The term *micro-coccus* or *coccus* is used either as a general term or to denote separate cocci, or cocci massed together in no regular fashion.

Such a classification as we have described can only be used for convenience, and by no means represents a natural classification. For a natural classification many other considerations have to be taken into account, such as the mode of growth, motility, chemical products, pathogenic properties, and so on.

It is often difficult to say whether an oval bacterium should be more properly called a bacillus or a coccus. Friedlaender's pneumonia bacillus, for example, is sometimes called a coccus and sometimes a bacillus. Many bacteria—such, for instance, as the bacterium of fowl cholera—are remarkably pleomorphic; and even in the same colony we may meet with bacilli forms and cocci forms side by side.

Again the morphology of most bacteria varies according to the mode of cultivation. The bacillus pyocyaneus, when grown on most media, takes the form of short, straight rods, but by cultivation in special media corkscrew-shaped bodies are produced.

In classifying the bacteria according to their shape all we can do is to indicate which form is the most predominant. For example, the pneumococcus is usually called a diplococcus; but under certain circumstances long chains are formed, and it would then come more correctly under the designation of streptococcus.

CULTIVATION OF BACTERIA.

Bacteria can be cultivated upon a variety of media, both fluid and solid. The most common fluid medium is broth made from beef or veal to which peptone and salt is generally added. Of the solid media broth stiffened by the addition of gelatine or agar is much used, the gelatine for cultivation at low, and the agar at higher temperatures. Coagulated blood serum is also an excellent medium for the growth of many bacteria.

These media are placed in test-tubes plugged with cotton wool, and are then sterilised by steam. The cotton wool prevents the germs in the air from gaining access to the interior of the tubes; and the tube, when once sterilised, will remain uncontaminated for an indefinite period.

Inoculation of the tubes is performed in the following way. A little of the material we wish to transplant is taken up by means of a platinum wire or loop fixed in a glass rod, and previously sterilised by heating in the flame. The cotton wool plug is then removed from the

tube, and the wire either streaked over the surface of the medium (streak cultivation), or is plunged into the depth (stab cultivation), or, if the medium is fluid, the loop is dipped into the medium.

Under suitable conditions of temperature the newly sown bacteria rapidly multiply, and on solid media form colonies visible to the naked eye. The shape, size, colour, and mode of growth of the colonies are often characteristic, and afford an important aid in determining the species.

Gelatine and agar tubes can be used for making plate cultivations. The medium is melted, when sufficiently cool it is inoculated, and before it has set it is poured out into a shallow sterile dish provided with a cover, so as to form a thin layer. When colonies have developed they can be examined directly under the low power of the microscope, and then often present characteristic appearances.

Pure cultivations from a mixture of colonies of different bacteria can be obtained by transplanting from the individual colonies into tubes. Thus by means of plate cultivations a mixture of bacteria can be separated, and streak cultivations can often be used with the same object.

CONDITIONS OF GROWTH OF BACTERIA.

As a rule, no growth occurs below 5° C. or above 50° C.; but certain species will grow at 0° C., and others at as high a temperature as 60° C.

When exposed to high temperatures the bacteria are destroyed, the thermal death point varying with the different species. When desiccated the bacteria will withstand much higher temperatures than when moist. In order to

destroy sporeless bacteria a temperature of $120^{\circ}\text{C}.$, acting for an hour and a half, is necessary, while spores require a temperature of $150^{\circ}\text{C}.$ for one hour. In the moist state sporeless pathogenic bacteria are destroyed by a temperature of $60^{\circ}\text{C}.$ acting for ten minutes.

Most spores are destroyed by steam or boiling water at $100^{\circ}\text{C}.$ in a few minutes; but for some species steam at $120^{\circ}\text{C}.$, acting for twenty minutes, is necessary.

All the pathogenic bacteria grow well at the temperature of the human body, and some grow at the ordinary temperature of the air, about $20^{\circ}\text{C}.$ For cultivating at higher temperatures an incubator is used, consisting of a large chamber with a water-jacket, heated by a gas-burner provided with a regulator which can be set at any temperature desired.

Most bacteria grow only in the presence of oxygen, but others only in its absence. The former are called *aerobic*, the latter *anaerobic*. Some, however, will grow either in the presence or in the absence of oxygen (facultative anaerobes).

The composition and reaction of the medium are important factors in determining growth. As a rule, the medium should be neutral or faintly alkaline.

Some bacteria, such as the leprosy bacillus, have not yet been cultivated, although attempts have been made upon a large variety of different media. Other bacteria will only grow on special media; the tubercle bacillus, for instance, will grow only upon agar when glycerine is added.

The addition of many chemical substances to the medium prevents the growth of bacteria. The following table shows the results of careful experiments made by Dr. Percy Evans in the bacteriological laboratory of Guy's

Hospital, and recorded by him in Vol. XLVII. of the Guy's Hospital Reports.

Chemical.	Strength of Solution in broth sufficient to prevent growth of	
	Anthrax bacillus.	Staphyloc. Pyogen. Aureus.
Corrosive Sublimate	1 : 70,000	1 : 50,000
Carbolic Acid	1 : 500	1 : 300
Creolin	1 : 5000	1 : 2500
Coal-tar Emulsion	1 : 5000	1 : 2000
Oil of Sanitas	1 : 4000	1 : 1000
Ol. Pini Silvestris	1 : 4000	

In order to actually kill bacteria more powerful solutions are required. The following table shows some of Dr. Evans' results.

BACTERIA DESTROYED.

ANTHRAX BACILLI.			ANTHRAX SPORES.		STAPHYLOC. PYOGEN. AUREUS.	
	Strength of Solution.	Time.	Strength of Solution.	Time.	Strength of Solution.	Time.
Corrosive Sublimate,	1 : 25,000,	$\frac{1}{2}$ hr.	1 : 3000	1 hr.	1 : 200	$\frac{1}{4}$ hr.
"	1 : 15,000,	1 min.	1 : 1000	$\frac{1}{4}$ hr.		
Carbolic Acid . .	1 : 150	$\frac{1}{4}$ hr.	1 : 20	24 hrs.	1 : 50	$\frac{1}{4}$ hr.
" . . .	1 : 100	5 min.				
Creolin . . .	1 : 300	1 min.	1 : 10	no effect in 2 days.	1 : 75	$\frac{1}{4}$ hr.
					1 : 100	24 hrs.
Coal-tar Emulsion	1 : 300	1 min.	1 : 10	no effect in 2 days.	1 : 40	no effect in 24 hrs.
(Liq. picis carbonis)						
Oil of Sanitas .	1 : 200	$\frac{1}{4}$ hr.	1 : 5	no effect in 24 hrs.	1 : 20	no effect in 24 hrs.

Exposure to *direct sunlight* prevents the growth of bacteria, and prolonged exposure causes death.

STAINING OF BACTERIA.

Bacteria are best stained by the aniline dyes. Those most commonly used are methylene blue, fuchsin, and gentian violet, dissolved either in plain water or in an aqueous solution of carbolic acid or aniline oil.* Cover-glass preparations are made in the following way. The material is spread in a thin film on a cover-glass by

* For formula see Appendix.

means of a platinum wire. If we are examining pus or tissue-juices it is sufficient to smear the material directly over the cover-glass ; but with colonies on solid media a drop of water must be first placed on the cover-glass. The film is allowed to dry in the air, and is then heated by passing the cover-glass three times through the flame of a spirit lamp. The object of this procedure is to fix the film upon the cover-glass, and to render the albuminous material transparent. The cover-glass is then immersed for several minutes in the stain, washed in water, dried, and mounted in Canada balsam.

In staining by Gram's method the cover-glass is prepared in the usual way, and is then placed in a solution of gentian violet in aniline water.* After remaining in this solution for about five minutes it is placed in a solution of iodine (the liquor iodi of the Pharmacopœia diluted to a light sherry colour). It is kept in this solution until the film becomes dark brown ; it is then decolorised with alcohol, washed in water, dried, and mounted in Canada balsam. Certain bacteria are decolorised by this method, while others are stained of a deep purple colour. This method of staining is consequently of some diagnostic value.

Tubercle bacilli are stained by a special method. The cover-glass is stained in a warm solution of fuchsin in carbolic acid,* and is then dipped in a 25 per cent. solution of sulphuric acid, washed in water, and mounted in Canada balsam. The sulphuric acid removes the stain from all bacteria with the exception of the tubercle and leprosy bacillus, and consequently this method of staining is of diagnostic value. In staining sputum methylene blue may be used as a counterstain after

* For formula see Appendix.

decolorising in the acid ; the tubercle bacilli then appear red, and the nuclei of the cells and any other bacteria present blue.

MOTILITY.

Many bacteria are motile, and when examined under the microscope in the living state can be observed to move about from place to place, either with rapid darting or with slow undulatory movements. These bacteria are provided with one or more thread-like appendages, called flagella, which can be demonstrated by special methods of staining.*

CHEMICAL PRODUCTS OF BACTERIA. TOXINES.

The bacteria produce a number of chemical changes in the media in which they grow. These changes are of two kinds: the one is the elaboration of bodies of a more or less complex nature, the other is the splitting up of complex molecules into simple bodies, such as carbonic acid and sulphuretted hydrogen. The two processes, the building up and the breaking down, go on at the same time.

All bacteria during their growth elaborate from the medium the protoplasm of which they are composed. The principal chemical constituent of the protoplasm is a substance called *protein*. This substance appears to be the same for all kinds of bacteria. Some bacteria are able to elaborate their protoplasm out of simple organic bodies such as tartrates, while others require more complex bodies as proteids. Besides protein other complex bodies are elaborated, which serve various purposes in the economy of the bacteria. Many bacteria produce *pigments*,

* For formula see Appendix.

which give a characteristic colour to the colonies or to the medium in which they grow. Others produce *ferments*. One of these, which is frequently met with, liquefies gelatine, converting it into a form of peptone. This power of liquefying gelatine is of great value for the purpose of differentiating the species.

The most important substances produced by bacteria from the point of view of pathology are various poisonous bodies called *toxines*. The exact chemical nature of many of these bodies has not yet been determined. Some are *alkaloids*, and can be obtained in a crystalline state. But still more important, on account of their extremely poisonous nature, are the so-called *toxalbumens*. It is probable that they are allied to ferments, but, whether this is so or not, they have never been obtained in a state at all approaching purity. The toxic properties of some of these bodies is extraordinary; an almost imponderable quantity of the tetanus toxine, for instance, being capable of producing fatal tetanus in a mouse.

The toxines produced by the pathogenic bacteria are of a specific character, and when injected give rise in animals to symptoms similar to those caused by the bacteria from which they have originated. They are very unstable bodies, and rapidly decompose. The different toxines possess different physiological actions. The majority cause pyrexia, but some, such as the toxine of cholera, cause a rapid fall of temperature. Many cause effusions, petechiæ, and hæmorrhages in various parts of the body. Some cause paralysis, others spasm of the muscles, and others diarrhœa. The same bacterium may produce several different toxines.

The bacterial protein is also poisonous, but to nothing like the same degree as the toxines we have just de-

scribed. Nor is it specific, for all species of bacteria appear to produce the same protein.

PATHOGENIC PROPERTIES OF BACTERIA.

Some bacteria, when introduced into the tissues of a living animal, are rapidly destroyed without producing any ill effects. Such bacteria are called *Saprophytes* (*σαπρός*, putrid, *φυτόν*, a plant; these organisms are frequently found in decaying or putrifying organic matter). Others are capable of multiplying within the tissues of animals and of producing various inflammatory and other changes, which often lead to a fatal termination. They are called true *Parasites* or pathogenic bacteria, and are divided into two classes—the *strict* and the *facultative* parasites. The former, of which we may give the leprosy bacillus as an example, do not appear to be capable of leading an independent existence outside the body; while the latter, of which a good instance is the anthrax bacillus, can multiply outside the body, and, in fact, often lead a saprophytic in preference to a parasitic life. Many of the parasitic bacteria, although incapable of growing outside the body under the usual conditions of nature, yet can be artificially cultivated on various media. Again, many bacteria retain their vitality for a long time outside the body, although under this condition they are not capable of multiplying.

The susceptibility of animals to infection with different bacteria varies enormously. Rabbits, for instance, when inoculated with the pneumo-coccus rapidly succumb to a general infection, the blood and tissues before death containing myriads of cocci; while fowls enjoy perfect immunity to this micro-organism. Guinea-pigs are more susceptible to the tubercle bacillus than rabbits, while the converse is

true in the case of the pneumo-coccus. As a general rule, young animals are more susceptible to infection than older ones.

The virulence of any pathogenic bacterium also varies according to the manner in which it has been cultivated. By cultivation at relatively high temperatures—*e.g.*, 40°-45° C.—by protracted cultivation on artificial media, or by the action of various chemical substances, the virulence can be attenuated to such an extent that what was originally a highly virulent bacterium can be rendered almost, if not entirely, harmless. When virulence has once been lost it is a difficult matter to restore it, but this can be done by repeated passages through the bodies of animals.

MODE OF ACTION OF PATHOGENIC BACTERIA.

Bacterial diseases have been divided into two classes, *toxic* and *septic*. In *toxic* diseases the bacteria multiply only at the spot of inoculation, where they produce toxins which are absorbed, and cause the symptoms of the disease by their injurious effects upon the tissues. A good example of this class of disease is that produced in the guinea-pig by inoculation with the diphtheria bacillus. That the ill effects are due to the toxins is shown by the fact that exactly similar symptoms can be produced by injecting the toxins from which the bacilli have been freed by filtration. In *septic* diseases the bacteria multiply not only at the spot of inoculation, but also in the blood and tissues. As an example of this form of disease we may quote the disease produced in rabbits by inoculation with the pneumo-coccus. Between these two forms, the toxic and the septic, are diseases which occupy a transitional place.

ENTRANCE OF BACTERIA INTO THE BODY.

Animals can be artificially inoculated with pathogenic bacteria in a variety of ways. Subcutaneous inoculation is the method usually employed, but infection can be obtained by injection into the peritoneal cavity, the muscles, joints, or the circulatory system; and in some cases through the lungs by inhalation, or through the intestines by feeding. The skin offers considerable resistance to the entrance of bacteria; nevertheless, infection has been produced by rubbing into the skin emulsions of bacteria. Many bacteria are destroyed by the hydrochloric acid of the gastric juice, so that infection through the digestive tract is possible only when the juice is artificially neutralised or its secretion deranged.

IMMUNITY.

We have already stated that some animals enjoy immunity to infection with bacteria which are pathogenic to other animals. This form of immunity is called *natural immunity*, and must be distinguished from *acquired immunity*. It must be remembered that immunity, whether natural or acquired, is to a great extent a relative matter, and depends upon the number and virulence of the bacteria introduced. An animal may not be affected when a few bacteria are introduced into its body, but will succumb if a large number are introduced.

Natural Immunity.—This depends upon a number of factors, the body possessing various means of defence against the invasion by bacteria. In some instances it is merely a question of body temperature. Frogs, for example, are immune to infection with tubercle, because the temperature of the frog is unsuitable for the growth of the

tubercle bacillus. In some instances it is a question of tolerance of the tissues to poison. Fowls enjoy immunity to infection with tetanus, because their tissues are not affected by the tetanus toxine, just as they can withstand large doses of morphia.

But in most instances immunity is due to the power possessed by the body of destroying the bacteria introduced. The destruction of the bacteria is brought about either by certain cells called *phagocytes*, or by the *fluids* of the body, or by a combination of these two factors, in some cases the one, in some cases the other, playing the predominant part. The phagocytes consist of certain kinds of leucocytes, and of endothelial cells of the blood-vessels and lymphatics, which can be observed to take up the bacteria, and destroy them by a process of digestion. The destruction of bacteria by the fluids of the body can be demonstrated by subjecting them to the action of the blood serum *in vitro*.

Falling short of actual destruction the bacteria may be hindered in their growth and in the production of toxins by the cells and fluids, and may be shut off from the rest of the body by an accumulation of cells. In such foci the bacteria may remain living for a long period, and should the resistance of the body be lowered they may start again into activity and give rise to a general infection.

Natural immunity may be weakened by various influences which lower the vitality of the body. Fowls, which are naturally immune to anthrax, can be rendered susceptible by exposure to cold; and the administration of drugs or exposure to fatigue has been shown to render animals, otherwise immune, susceptible to bacterial infection.

Acquired Immunity.—Susceptible animals can be rendered immune towards pathogenic bacteria in various ways. The main principle consists in inducing a mild form of the disease. This can be brought about by introducing minute, non-fatal doses of living bacteria, by inoculating with attenuated cultivations, or by injecting first small, and then larger doses of the bacterial toxines. The immunity takes some short time to be established, the time varying with the species of bacterium. When once established it lasts for some months only, and then disappears. Immunity produced in this way is, generally speaking, specific; that is to say, an animal which has been immunised against any pathogenic bacterium is only rendered immune towards that particular bacterium, and is not protected against inoculation with other bacteria.

A less durable form of immunity can be acquired by the previous injection of various substances. For instance, the injection of bacterial products of various kinds, and even the simple injection of broth into the peritoneal cavity of guinea-pigs, will protect them for a few hours against inoculation with virulent cholera vibrios.

The explanation of acquired immunity is by no means a simple matter, especially as it differs in different cases.

Some forms of acquired immunity—for instance, the temporary immunity we have just described—depend upon a reinforcement of the factors concerned in natural immunity, an increased phagocytosis, and an increased bactericidal power of the fluids of the body. But in most cases there is added an entirely new factor, which is not found in natural immunity. We are referring to the presence of protective substances in the fluids, especially the blood serum, of artificially immunised animals. When

the blood serum of an animal, rendered highly immune to any bacterium, is injected into an otherwise susceptible animal, it will protect it against infection with the same bacterium. And this protection is afforded whether the attempt at infection (usually by inoculation) is made subsequent to, at the same time as, or even before the injection of the serum.

In the toxic diseases, such as tetanus and diphtheria, the protective power of the serum is due to its antitoxic properties, its power of annulling the effect of the toxins. A minute quantity of the blood serum of an animal highly immunised to tetanus or diphtheria, when injected together with a fatal dose of the tetanus or diphtheria toxine into a susceptible animal, quite annuls the effect of the toxine. Hence the protective substances in the serum are called *antitoxines*. The antitoxines are of therapeutic value, for they will protect when injected subsequently to the introduction of the toxins. The antitoxines are, to a great extent, specific; the diphtheria antitoxine will not protect against tetanus, nor the tetanus antitoxine against diphtheria.

In septic diseases it appears that the protective substances of the serum act in combination with the fluids of the body in destroying the bacteria.

The foregoing considerations enable us to explain many points in connection with contagion. We have learnt that some animals are naturally immune to bacteria which are pathogenic to other animals, and that even a mild attack of some bacterial diseases protects against a second attack. The same is found to be the case with the specific fevers in the human subject, and no doubt the same explanations are applicable. Some individuals appear to be incapable of contracting certain specific fevers, even when they are

exposed time after time. Such an immunity is often similar to the natural immunity of animals, the contagion as soon as it enters the body being destroyed by the bactericidal properties of the cells and fluids. In other cases it is an artificial immunity induced by a previous mild unrecognised attack of the disease. It is well known that those in attendance upon cases of scarlet fever are liable to attacks of sore throat without developing the characteristic symptoms of scarlet fever. It is probable that many of these sore throats are really mild attacks of scarlet fever, which protect against a characteristic attack of the disease.

Immunity, whether natural or acquired, may be lost by any influences lowering the vitality of the tissues. A person in good health may be frequently exposed without harm to the contagion of any specific fever, but at once contracts the disease on exposure when out of health. We have already seen that the immunity of animals can be experimentally lowered by influences which lower the vitality of the body.

Some individuals appear to be extremely susceptible to all specific fevers, and contract almost every infectious disease to which they are exposed. We can only suppose that the bactericidal properties of the cells and fluids of their bodies are exceedingly ill developed.

An attack of a specific fever usually protects against a second attack, and this is doubtless due, to a great extent, to the formation of protective substances in the blood and other fluids, just as is the case in experimental bacterial diseases. Indeed, in diphtheria in the human subject, protective substances have been found in the blood after recovery. These protective substances do not remain indefinitely in the body, but are ultimately

eliminated, the length of time that elapses before elimination is complete varying according to the nature of the disease and many other circumstances. It is thus that we can explain the occurrence of second and even third attacks of the specific fevers.

The phenomena of recovery from an infectious disease are exceedingly interesting. They differ, at any rate in degree, in the toxic and septic diseases. Let us take as an example of the former class, diphtheria. Recovery here appears to be due to the formation of antitoxic substances in the body, which protect the tissues against the action of the toxins. At the same time the local inflammation at the spot of inoculation forms a barrier to the general invasion of the body by the bacilli. Any bacilli that get into the general circulation or the lymph stream are rapidly destroyed by the cells and fluids. But the bacilli are not so readily destroyed at the spot of inoculation, and may remain living and active for a long time after the patient has apparently recovered. During this time the toxins produced by the bacilli are annulled by the antitoxic substances in the body, so that the bacilli, as far as the patient is concerned, are in the position of harmless saprophytes, and are ultimately destroyed by the cells and fluid secretions. But should the acquired tolerance to the poison be in any way weakened, then the bacilli in reference to the patient become pathogenic, and a relapse occurs resembling, though generally milder than, the original attack.

In septic diseases, such as erysipelas, the chief effort of the body is to prevent the bacteria from invading the tissues generally. The cells and fluids prevent the growth of the bacteria, and ultimately destroy them. Probably antitoxic substances are also formed, but to a less extent

than in the toxic diseases. For some time after recovery the bacteria remain living, but are prevented from multiplying by being shut up in the cells. Should any depressing influence arise they become set free, and a relapse occurs.

CONCURRENCE OF FEVERS.

From the knowledge that one attack of a specific fever protects against a second attack, we naturally ask whether one specific fever will protect against another specific fever, either occurring at the same time or subsequently. We must answer both of these questions in the negative. Clinical observation gives no support to the view that one specific fever in any way protects against a subsequent attack of another fever; in fact, it is just the reverse. A patient during convalescence from one fever is more liable, if exposed, to contract another fever. Convalescents from scarlet fever and measles are more susceptible than healthy individuals to diphtheria; and attacks of measles and whooping cough frequently follow one another in the same patient. The severity of the second disease is usually greater than would be the case if the patient had not been previously ill. There can be no doubt that an attack of any specific fever lowers the general resisting powers of the body, and hence the importance of not exposing the patient to other infection during convalescence. Two or more specific fevers may run their course concurrently in the same patient without either being altered in character by the presence of the other. Thus a boy, aged four years, was attacked with diphtheria on April 30th. The illness was severe, and was followed by paralysis, from which he was still suffering on July 1st, when he was seized with symptoms of morbilli, and on July 8th

with scarlet fever. He made a good recovery. A boy, aged six years, suffering from varicella, was sent to a small-pox hospital. Being considered to be a doubtful case, he was vaccinated on the day of admission. The vaccination was successful; but twelve days after admission symptoms of variola appeared: the attack was much modified. Another boy, aged four years, was admitted to the Eastern Hospital with a mild attack of scarlet fever (rash, sore throat, pyrexia, and strawberry tongue), he had a well-marked varicella eruption, and was at the same time in the paroxysmal stage of whooping cough.

One of the reasons why two or more specific fevers do not more frequently occur together in the same patient is that a patient, when attacked by an infectious disease, is isolated, and is therefore kept from exposure to other infections.

THE MODE OF CONVEYANCE AND ENTRANCE OF THE CONTAGIA OF THE SPECIFIC FEVERS.

We have already seen that bacteria may be introduced into the body in various ways, and the same is true with regard to the contagia of the specific fevers. Most contagia have a predilection for attacking certain parts of the body. For instance, the virus of enteric fever appears to be only capable of primarily attacking the intestinal tract, and it is doubtful if it ever enters the body by any other channel. Other contagia, such as that of erysipelas, may enter the body in various ways, through the skin or the mucous membrane.

But the mode of entrance depends to a great extent upon the mode of conveyance, and this again upon the nature of the contagium.

In order for micro-organisms to be conveyed through the air they must be in a dry state, but some micro-organisms, such as the cholera vibrio, rapidly die when deprived of moisture, and thus the contagium of this disease is not conveyed by means of the air. The contagia of scarlet fever, measles, variola, varicella, and typhus fever appear to resist drying, and in consequence conveyance through the air is common.

Some bacteria, on account of their minuteness, are more readily conveyed by the air than others. The influenza bacillus is exceedingly minute, while anthrax spores are large, and thus the former is more readily conveyed through the air than the latter.

In order for contagion to enter through the digestive tract, it must be of such a nature that it can pass through the stomach without being destroyed by the gastric juice. We cannot rely entirely upon the results of experiments conducted upon healthy gastric juice, because in conditions of ill health the juice may either not be secreted in proper quantity or it may be deficient in quality. The fauces are a frequent seat of invasion on account of their position at the entrance to the digestive and respiratory tracts, so that they are liable to be attacked by contagia conveyed by food or by the air.

Some micro-organisms do not appear to be capable of leading an independent existence outside the human body, and rapidly die when removed. This, for instance, is the case with the contagium of syphilis ; and consequently infection but rarely occurs except by the direct contact of individual and individual. Others apparently do not multiply outside the body, but will retain their vitality for a considerable time. The contagium of measles may be quoted as an example. As far as we know the infection

either spreads directly from patient to patient or through fomites. Others can multiply in various articles of food, in water, or in the soil. The virus of enteric fever comes into this category, and infection is generally conveyed by means of water or food.

Some micro-organisms, such as that of glanders, are pathogenic to animals as well as to man; in this disease infection is almost invariably acquired by contact with diseased animals.

PROPHYLAXIS.

It will only be possible to stamp out most of the infectious diseases when we possess an accurate knowledge of the life history of the micro-organisms which cause them. The problem must be approached in different ways, according to the nature of the contagium. In some cases, as in enteric fever, the contagium can lead an independent existence in the soil and in water, so that our efforts must be directed chiefly to the prevention of contamination of the water supply, and to the establishment of good sanitation. In other cases, such as typhus fever, the contagium does not, apparently, multiply outside the human body; thus careful isolation and disinfection of fomites are of most importance.

For the purpose of affording sanitary authorities and medical officers of health information as to the prevalence of infectious diseases, and to facilitate isolation, the *Infectious Diseases Notification Act* was passed in 1889. The adoption of this Act is compulsory for London, but for provincial towns and districts it is optional. Many sanitary authorities have adopted the Act.

The following diseases have to be notified under the Act :—

- Small-pox.
- Cholera.
- Diphtheria.
- Membranous Croup.
- Erysipelas.
- Scarlet Fever or Scarlatina.
- Typhus Fever.
- Enteric Fever, or Typhoid Fever.
- Relapsing Fever.
- Continued Fever.
- Puerperal Fever.

But a sanitary authority can, under certain conditions, add any other infectious disease, such as influenza and measles, to the above list.

Every medical practitioner “attending on or called in to visit the patient, shall forthwith, on becoming aware that the patient is suffering from an infectious disease to which this Act applies, send to the medical officer of health for the district a certificate,” in which are to be stated, amongst other matters, the name of the patient, the address or situation of the house in which he is living, and the nature of the infectious disease from which he is suffering.

The sanitary authority provides the medical practitioners of the district with a book of forms or certificates for the purpose of notification.

In London the Metropolitan Asylums Board is the body responsible for the provision of isolation accommodation, and for the arrangements connected with the removal of infectious patients. As there is still some ignorance

concerning the means by which the removal of an infectious patient can be obtained, we have placed in the Appendix the regulations of the Metropolitan Asylums Board on the subject.

One of the great difficulties in successful prophylaxis by isolation lies in the occurrence of mild cases which are unrecognised; but increased knowledge of the nature of contagion will render the recognition of these cases an easier task. As regards diphtheria, a bacteriological examination now enables us to recognise the mildest forms of the disease, and we have reason to think that when this method is more generally employed a decrease of the disease will occur. Unfortunately at present we know practically nothing about the real nature of the contagia of many of the infectious fevers, and thus mild cases escape recognition. All attempts to stamp out scarlet fever have at present failed, and we do not believe that success will be attained until improved methods of diagnosis are discovered.

A general improvement in hygienic conditions is an important factor in the prevention of infectious diseases, for epidemics are favoured by overcrowding, starvation, squalor, and bad sanitation. The decrease of some infectious diseases is, no doubt, due in large measure to improved hygienic conditions.

The question arises whether it is advisable to try to stamp out many of the common infectious diseases in which such attempts have hitherto failed. When an epidemic disease attacks a nation or community for the first time the mortality is known to be exceedingly high. This is probably due to the absence of hereditary protection, since the ancestors of the people have not suffered

from the disease. On these grounds it is urged that as there is not a reasonable prospect of complete success, attempts to abolish the particular disease are not advisable. With this view we cannot agree; for we believe that with a fuller knowledge and experience the time will come when infectious diseases will be eradicated.

CHAPTER III.

DISINFECTION.

IT has already been stated that certainly some, and probably all, infectious diseases are due to the invasion of the body by living micro-organisms. These micro-organisms are given off in the exhalations and excretions of the patient, and, adhering to articles of clothing and utensils for eating and drinking, may retain their vitality for a long time. Infection may be conveyed by such articles, which are then called fomites (*fomes*, touch-wood, tinder).

In order to prevent the spread of infectious diseases it is necessary to render free from infection not only the patient, but also all articles that have been brought in contact with him.

The object of disinfection is to destroy the micro-organisms which are the cause of the disease. The difference between a *disinfectant* and an *antiseptic* is that the former destroys germs and the latter only hinders their growth. This distinction is often forgotten, especially when chemical substances are employed for so-called disinfecting purposes. Many substances are excellent antiseptics but are practically of no value as disinfectants.

We will first speak of the disinfection of inanimate objects.

The most important disinfecting agents are (i) Heat ; (ii) Chemical substances ; (iii) Direct sunlight.

(i) *Heat* is applied either as *dry* or *moist* heat ; the latter is by far the most effective and reliable disinfectant we possess.

For the application of *dry heat* the articles are placed in a chamber heated either by gas or by an outer jacket containing steam at a high temperature. In order to destroy all micro-organisms the temperature should remain at 150°C . for an hour ; but at this temperature articles of clothing, etc., are injured if not actually destroyed. There are other objections to the use of dry heat. The temperature varies considerably in different parts of the disinfecting chamber, and the heat does not penetrate into the interior of such articles as pillows and mattresses. Consequently this method of disinfection should only be employed for articles which will not stand the application of moist heat, such as leather goods, boots, etc. The temperature of the chamber should be 110°C . (230°Fahr .), and should be maintained for at least two hours.

Moist heat is employed either by boiling in water or by exposure to steam. For disinfecting small metal articles, such as surgical instruments, tongue depressors, spoons, forks, etc., boiling in water for a quarter of an hour is a good method.

For disinfecting bulky articles, such as pillows, mattresses, and clothing, an apparatus into which steam under pressure can be introduced is absolutely necessary. The temperature of the steam should be 115°C ., corresponding to a pressure of 10 lbs. to the square inch, and the articles should be exposed for half an hour. The steam should

be *saturated* and not *superheated*. The difference between saturated and superheated steam is as follows. When water is heated in a boiler communicating with a closed chamber, the steam given off is saturated, and its temperature depends upon the pressure. Any lowering of the temperature of the steam causes condensation, and the same result would ensue if any additional external pressure were applied. If, however, steam from a boiler is conducted into a chamber, and this chamber is itself heated, the steam becomes superheated, and behaves in a manner different from saturated steam. In order to cause condensation of superheated steam the temperature must be considerably lowered, or a considerable external pressure must be applied. Superheated steam behaves like a gas, but saturated steam like a vapour.

Now it has been shown that superheated steam possesses all the disadvantages of dry heat for the purposes of disinfection, and hence its use should be discarded. In some forms of apparatus in which the steam under pressure is said to be superheated, the superheating is so slight that it may be neglected, and consequently such an apparatus is quite reliable and efficacious.

The great advantage of saturated steam is its penetrating power, which may be increased by intermitting the pressure several times during the process of disinfection, or by the use of a vacuum pump, which partially exhausts the chamber before the steam is introduced.

The apparatus should have two doors, each leading into different rooms; through one door the infected articles are placed in the chamber, and through the other they are taken out after disinfection. Thus the infected and disinfected articles are kept separate.

Some of the high-pressure machines are constructed

with an outer steam jacket, and can be employed for the application of dry as well as of moist heat.

Certain india-rubber goods, and articles in which glue is used, cannot be disinfected by heat in any form.

Letters can be disinfected by steam if they are not sealed by wax, and so may books, unless the binding is of leather. In the latter case the book should be taken out of its binding and disinfected by steam, and the binding by dry heat.

(ii) *Chemical Disinfectants* are employed either as *solutions* or in the *gaseous form* (*fumigation*). Certain chemicals have, doubtless, been shown to be good germ-destroyers by test-tube experiments. But for the disinfection of bulky articles their efficiency is far inferior to that of heat, especially of moist heat. Whether applied in solution or as gas, they do not penetrate readily into the depths of the articles to be disinfected. Corrosive sublimate, indeed, forms insoluble compounds with albuminoid substances, and these compounds, forming a layer upon the surface, protect the underlying portions. Again, the length of time which must be allowed for most of the chemical disinfectants to act, and the strength of solution in which they have to be applied, constitute grave objections to their sole use in practical disinfection. But they are useful in cases when it is requisite to disinfect only the exterior of objects, or where it is impossible to employ heat. The best and most commonly used chemical solutions are carbolic acid, of a strength of not less than 5 per cent., and corrosive sublimate (mercuric chloride), not less than 1 in 1000.

Of gaseous chemical disinfectants, sulphur dioxide is the one most frequently used, but its disinfecting action is very imperfect. It is usually employed for

fumigating the interior of closed spaces, such as rooms and carriages. The necessity of introducing plenty of fresh air into the room after fumigation is by no means an unimportant factor in the efficacy of the method. Fumigation is quite useless for disinfecting mattresses, clothes, and similar articles, however much they are exposed to the gaseous fumes.

Sulphur dioxide is generated by burning sulphur in an iron pot; it can also be obtained compressed in cylinders. Three pounds of sulphur are required for every 1000 cubic feet of air space.

When a room is to be fumigated it should be rendered as air-tight as possible. The chimney, windows, doors, and ventilators should be closed and pasted up with paper, and not opened until twenty-four hours after the commencement of the process of fumigation. In the case of sulphurous acid experiments have shown that its action is more certain when the air of the room is moist. Steam, therefore, should be allowed to escape into the air of the room for half an hour or so before the generation of the gas, or the walls, ceiling, and floor may be sprayed with water. After fumigation, the doors, ventilators, and windows should be thrown widely open, and kept so for at least two days.

The vapour of formic aldehyde is a most efficacious disinfectant, but is too expensive to be used on a large scale. Its use must be restricted to the disinfection of valuable articles such as furs, which are injured by exposure to heat. The best method for employing it is to place the articles into a small air-tight chamber, together with a beaker containing some of the liquid.

(iii) The germs of some of the infectious diseases

are rendered inert by prolonged *exposure to sunlight* and air. Therefore when no other means of disinfection is procurable, bedding, clothes, and other infected articles should be freely exposed for, at the very least, twenty-four hours to the sunlight and air.

The *disinfection of the patient* would be a matter of great difficulty were it not for the power possessed by the body to destroy the germs of disease. After a certain lapse of time, which varies for each disease (see Appendix), the patient ceases to be infectious, provided that during convalescence he has frequent baths, and is allowed to go out daily into the open air. Towards the end of the period of isolation the patient should be washed daily from head to foot with carbolic soap. Immediately before his discharge he should take a similar bath, if possible in a different room from that in which he has been kept isolated. He should then, wrapped in a large clean towel, go into another room, where clean clothes that have not been in contact with any infected articles should be prepared in readiness for him to put on.

Patients recovering from scarlet fever or diphtheria are the most difficult to disinfect; for in spite of all precautions such patients (especially the former) may remain infectious for weeks, and even months, after the usual period of isolation. The germs of the disease may escape the germicidal influences of the body, and may remain latent within the recesses of the nasal fossæ and behind the arches of the palate, and in the many depressions found in this region. In such situations they are protected from the action of antiseptic irrigations and sprays. Fortunately in the case of diphtheria the infectivity of the patient can be determined by a careful bacteriological examination. In scarlet fever this method

cannot be applied, but there can be little doubt that the scarlet fever germ possesses much tenacity of life, which renders the disinfection of the patient a very difficult matter.

We will now consider what precautions should be taken to prevent the spread of infection when a patient suffering from an infectious disease is treated at home.

The room in which the patient is isolated should be at the top of the house. Only the necessary articles of furniture should be left in the room. A sheet kept moist with a 1 in 40 solution of carbolic acid should be nailed in front of the door. Those nursing the patient should be kept isolated from the rest of the family, and should occupy rooms adjoining that of the patient. The medical attendant should wear a gown made of some washable material when visiting the patient, and should thoroughly cleanse and disinfect his hands in carbolic or sublimate lotion after each visit.

Soiled linen should be immediately placed in a 5 per cent. solution of carbolic acid, and should be washed by the nurses in attendance upon the patient. Pieces of rag which can be burned should be used in preference to pocket-handkerchiefs. Remnants of food and all dressings and bandages should be burned. The crockery used should be washed by the nurses in the sick room, or in an adjoining room.

Carbolic acid, or a solution composed of corrosive sublimate $\frac{1}{2}$ oz., hydrochloric acid 1 oz., water 3 gallons, should be added to the dejecta before they are thrown into the drains.

The patient should not be allowed to send away letters unless they have been disinfected by moist heat.

When an infectious patient has been removed, the

apartments he has been using and their contents should be thoroughly disinfected. All articles of little or no value should be burned. All personal linen and bed-linen should be soaked in 1 in 20 carbolic solution for several hours, then rinsed in cold water, and boiled before being washed. Bedding, blankets, clothes, curtains, carpets, and books should be disinfected by moist heat. This can be done by the sanitary authorities if they possess a reliable disinfecting apparatus, which is by no means always the case, even in London ; otherwise, there are private firms who perform disinfection most thoroughly. The furniture should be washed with carbolic acid or corrosive sublimate solution, as should all crockery-ware, india-rubber, and large metal articles. Small metal articles should be boiled or submitted to dry heat. Leather goods should be disinfected by dry heat. The paper should be stripped off the walls and burned. The rooms should be fumigated in the manner previously described. After fumigation the floor and walls should be thoroughly scrubbed with carbolic or sublimate solution, the ceiling sprayed with the same solution, the walls repapered, the ceiling whitewashed, and the woodwork repainted.

Similar precautions should be taken with respect to those who have been in constant attendance upon the patient ; their apartments, clothing, etc., should be disinfected.

CHAPTER IV.

ON RASHES SIMULATING THOSE OF THE SPECIFIC FEVERS.

I. **E**RYTHEMATA.—The rashes of some of the specific fevers belong to the class of erythematous rashes ; but similar erythematata may be produced by other causes. It is consequently impossible to diagnose a specific fever by the character of the eruption alone ; the other phenomena of the disease must be taken into account. A patient, for instance, may present a punctate red rash diffused over the trunk and limbs, and yet not be the subject of scarlet fever ; or he may present a maculated eruption having the character and distribution of that of measles, and yet not be suffering from that disease.

Certain *drugs* produce erythematous rashes. The character of these rashes is variable. The same drug may produce on different occasions a scarlatiniform, or a morbilliform rash, or a combination of these two varieties of erythema. Frequently, also, there is urticaria.

The outcome of these erythematous rashes is often accompanied by a rise of temperature, and the fauces may be reddened. Branny desquamation may follow ; sometimes the skin peels off in large flakes. Usually, but not

invariably, the desquamation is limited to the part affected by the rash.

Dr. Crocker enumerates thirty-five drugs which have been known to produce rashes of various kinds. But we are only concerned here with those drugs which, with any frequency, produce rashes such as are seen in the specific fevers. The chief drugs producing erythemata are *copaiba*, *belladonna*, *morphia*, *quinine*, and the antitoxic sera for diphtheria and tetanus. The bromides and iodides cause papular and pustular eruptions.

Copaiba gives rise usually to slightly raised red patches, situated chiefly upon the trunk and extremities, but sometimes on the face; the patches may remain discrete, or may run together to form large irregularly shaped erythematous areas. Urticaria is common. Very rarely *copaiba* produces a scarlatiniform rash. Occasionally the rash is hæmorrhagic. The odour of the drug may be detected in the urine, from which the resin may be precipitated by nitric acid.

The rash of *belladonna* is usually a diffuse blush, but sometimes it is punctate like that of scarlet fever. It has been observed most often in cases where a poisonous dose of the drug has been taken, but it has sometimes followed medicinal doses, especially in children, and even the application of a *belladonna* plaister. The temperature is raised, the pulse rate is increased in frequency, the pupils are dilated, the mouth and throat are dry, and there is active delirium.

There will be no difficulty in making a diagnosis in cases where the fact of the recent administration of these drugs is known. Apart from such knowledge, careful attention should be paid to the presence or absence of other symptoms.

Morphia, when taken by the mouth or given in a suppository, may produce a scarlatiniform, or a morbilliform rash. Hypodermic injections of the drug are not followed by rashes.

Quinine, when producing a rash, usually gives rise to a scarlatiniform erythema; the face, limbs, and trunk may be affected. There is often vomiting and pyrexia, and the rash is attended with severe itching. The rash is usually marked off from the unaffected skin by a well-defined margin. Desquamation follows, and may last for three or four weeks. Occasionally quinine produces a measly eruption.

The *diphtheria* and *tetanus antitoxic sera* produce, besides erythema multiforme and urticaria, a rash which is papular, blotchy, and exceedingly like that of measles. It often invades the whole of the skin. Sometimes there is puffiness of the face and injection of the conjunctival vessels. The faucial mucous membrane may be reddened and swollen. Very occasionally the rash is scarlatiniform; even then the face may be affected. Desquamation, often profuse, follows. These rashes appear from five to twenty days after the injection of the serum. The rash frequently begins to appear at or near the seat of injection—an important point to note in cases when it is a question of rubeola, measles, or scarlet fever on the one hand, against a drug rash on the other. There is often pyrexia, but no vomiting, coughing, sneezing, or bronchitis. In the scarlatiniform variety the sore throat, if present, is slight; there is no circum-oral ring; and the rash often affects large areas of skin, these areas being bounded by a well-defined margin. The condition of the tongue is not like that usually met with in scarlet fever. The desquamation is not "ringed." *Tuberculin* has been observed to give rise to similar rashes.

Iodide of potassium occasionally produces a blotchy

or a scarlatiniform erythema. There may then be injection of the conjunctival vessels and coryza.

The following conditions are often accompanied by rashes very similar to those of measles, rubeola, and scarlet fever—*septicæmia*, *uræmia*, and less commonly *acute rheumatism*. A rash may be the most prominent symptom of septic or uræmic poisoning. The morbilliform variety is the most common. There is no bronchitis, coryza, or sneezing. There may be pyrexia. In fact, the remarks that have been made concerning the rashes produced by antitoxic serum apply to many of the cases of septic and uræmic rashes. These rashes are described by dermatologists as *Erythema Scarlatiniforme* and *E. Morbilliforme*.

Two other rashes are occasionally mistaken for that of scarlet fever, *Erythema fugax* and *Idiopathic roseola*.

In *Erythema fugax* transient patches of redness appear on the face and trunk. There are no other symptoms of scarlatina.

In *Idiopathic roseola* (*Erythema roseola*) the rash may be local or general; it may consist of irregular patches of redness, or it may be slightly papular; it may last several days, disappearing from one place to appear in another. The palate is sometimes reddened. There is transitory pyrexia and restlessness. It is most often met with in children. The patchiness of the rash, its disappearance and reappearance, and the absence of sore throat and other symptoms of scarlet fever, will usually distinguish it from that disease.

2. PAPULAR AND PUSTULAR ERUPTIONS.—The *bromide* and *iodide* of potassium produce most commonly papular and pustular eruptions, more likely to be mistaken for variola than any other specific fever. For the diagnosis the reader is referred to the chapter on Small-pox.

CHAPTER V.

ON SORE THROAT.

THERE are several affections of the throat which are not infrequently mistaken for scarlet fever or diphtheria, especially the latter. It is thought, therefore, that a brief description of these affections will be useful to the student.

1. CATARRHAL SORE THROAT.—The tonsils, soft palate and its pillars, and mucous membrane of pharynx, are reddened and slightly swollen, and, later, mucus is secreted in excess. The patient complains of dryness of the throat, with discomfort or pain in swallowing. There may be slight febrile symptoms. The catarrhal inflammation often affects the larynx, trachea, and bronchi, giving rise to hoarseness, cough, and pain in the chest. The nasal passages may also be involved. A common predisposing cause of this form of sore throat is exposure to wet and cold.

2. ULCERATED SORE THROAT.—One or both tonsils are swollen and superficially ulcerated, and there may be ulcers on the soft palate or pharynx. The cervical lymph-glands may be enlarged. There is much pain in swallowing. The patient feels ill, and the temperature is raised. This form of sore throat results from exposure

to bad hygienic conditions, sewer-gas, the emanations from septic wounds, etc. It was at one time common amongst medical students, nurses, and others in attendance upon the sick in hospitals, and was known as "hospital sore throat."

3. SIMPLE ACUTE TONSILLITIS.—One or both tonsils are swollen, reddened, and painful. The surface may be covered with exudation, which is sometimes distinctly membranous. The symptoms are similar to those met with in ulcerated sore throat. Amongst the causes are bad hygienic conditions and exposure to cold. In many cases salicylate of soda is of great benefit. Simple tonsillitis is not infrequently met with in cases of rheumatic fever. This form of sore throat has, indeed, been described as "rheumatic sore throat."

4. QUINSY OR SUPPURATIVE TONSILLITIS.—This affection is almost entirely confined to adults. The tonsil on one side and the neighbouring structures become reddened and swollen. Great pain is experienced in swallowing, and there is marked pyrexia and febrile symptoms. The inflammation usually terminates by the formation of pus. Though the disease is, as a rule, unilateral, nevertheless both sides may be affected, one after the other.

5. FOLLICULAR OR LACUNAR TONSILLITIS.—Both tonsils are swollen, painful, and slightly redder than normal. The follicles are distended with exudation, and appear as yellow projecting spots upon the surface of the tonsils. The exudation often spreads upon the surface so as to form a thin layer, and the tonsils are then covered with patches that look like membrane. There is, as a rule, only moderate constitutional disturbance.

6. HERPES OF THE PALATE AND TONSILS.—Vesicles

come out upon the mucous membrane. They appear as reddish spots, or as whitish or grey spots surrounded by a red zone. Sometimes the spots consist of small superficial ulcers. They may be discrete, or coalesce to form irregular patches. Perfect vesicles are rarely observed. There is slight inflammation of the surrounding mucous membrane. Herpetic vesicles very often appear simultaneously about the lips, or on the face or ear. The attack may commence with a rigor, and there are moderate febrile symptoms.

7. ERYSIPELAS OF THE FAUCES.—In this affection there is extreme brawny swelling of the mucous membrane and submucous tissue. The surface of the mucous membrane is of a dark red, sometimes purple, hue. There is much pain in swallowing. Thick tenacious mucus collects; occasionally a membranous exudation forms. The affection, which is very fatal, appears sometimes to be dependent upon diseased teeth. The constitutional symptoms are those of a severe fever.

8. SYPHILITIC SORE THROAT.—In the secondary stage of syphilis sore throat is common. The mucous membrane of the palate and its arches and of the pharynx becomes more red than usual, and may be swollen. The tonsils are also inflamed, and shallow excoriations form upon their surface. Greyish, slightly elevated patches, and ulcers with a yellowish base and a bright red margin, may also appear upon the fauces. White spots (*plaques opalines*) are not infrequently seen on the mucous membrane of the fauces, hard palate, mouth, and lips. During the late (tertiary) stage of the disease deep and destructive ulceration of the soft palate may occur.

9. In TUBERCULOUS ULCERATION the floor of the ulcer presents pale, yellowish, coarse granulations, from

which much secretion is poured out. The ulcer, though not deep, may be extensive. This form of ulceration is found in those who are the subjects of pulmonary phthisis or of lupus. There is little pain ; the ulceration begins insidiously and runs a slow course.

10. For GANGRENE OF THE FAUCES see the chapter on Diphtheria.

The differential diagnosis of these diseases from scarlet fever and diphtheria is discussed in the chapters which deal with those affections. It may, however, be said here, that the diagnosis of diphtheria, and less often of scarlet fever, from ulcerated sore throat, simple acute tonsillitis, and follicular tonsillitis, is at times very difficult. Another point which should be borne in mind is that the quality of infectiousness is by no means confined to scarlet fever and diphtheria. Epidemics and outbreaks of sore throat have been described which have certainly not been due to either of these two diseases ; and in these outbreaks the forms of sore throat observed have been usually simple tonsillitis, ulcerated sore throat, and follicular tonsillitis.

CHAPTER VI.

SCARLET FEVER OR SCARLATINA.

IN this disease the principal symptoms are fever, a punctate erythematous rash followed by desquamation, inflammation of the fauces, and a liability to certain complications, of which the most important is nephritis.

Etiology.—The *geographical distribution* of scarlet fever is wide. It extends throughout the north-western countries of Europe, Russia, and, to a less degree, Italy, Turkey, Greece, and some of the Mediterranean islands. It is also met with in North, and parts of South America, but it is infrequent in Australia, Asia, and Africa.

With respect to *season*, in England scarlet fever is most prevalent during the last quarter of the year, and during this period the mortality (per 1,000 living) is highest. The minimum number of cases and of deaths occurs in the spring (April); during the summer the numbers gradually rise, and reach a maximum in October; while a fall begins in December, and continues to the minimum in April.

There is some evidence to show that the *fatality* (case mortality) is lowest when the disease is most prevalent, and *vice versâ*.

Scarlet fever is *disseminated* in several ways. It undoubtedly spreads from person to person, and can also be conveyed by means of fomites. The virus is not very diffusible, but it is very tenacious of life, infected garments that have been put aside for months having been known to originate an outbreak of the disease. The virus appears to be given off in the breath, in the secretions from the nose, mouth, pharynx, and ears, in the desquamating skin, and perhaps by the kidneys.

There is no evidence that the poison of scarlet fever has ever been conveyed by means of the water supply. The milk supply, however, has on several occasions been shown to be answerable for local outbreaks. In some instances a direct contamination of the milk by persons suffering from scarlet fever has been established. In others no such infection could be traced even after the most careful investigation, but the cows supplying the milk were found to be suffering from a disease of the udders, skin, and viscera; and it has been supposed that this was really scarlet fever in a modified form.

There were 36,849 cases of scarlet fever notified in London during 1893, with 1,599 deaths. This was a case rate per 1,000 inhabitants of 8·6, and a fatality of 4·34 per cent. For 1891 and 1892 the case rate was 2·7 and 6·4 per 1000, and the fatality 5·1 and 4·3 per cent. For 1894 the case rate was about 4·2 per 1,000, and the fatality 5·2 per cent.

When *age* is considered, we find that scarlet fever is almost entirely a disease of childhood and young adult life. This is shown in the following table, which gives the number of admissions and deaths at various ages

in the Hospitals of the Metropolitan Asylums Board from 1871 to 1894, together with the percentage fatality.

	Cases admitted.	Deaths.	Fatality per cent.
Under 5	23,072	4,052	17·6
5 to 10	33,647	1,789	5·3
10 „ 15	14,399	345	2·4
15 „ 20	5,319	139	2·6
20 „ 25	2,509	65	2·6
25 „ 30	1,215	38	3·1
30 „ 35	665	31	4·7
35 „ 40	281	16	5·7
40 and upwards	243	15	6·2
	<hr/>	<hr/>	<hr/>
Total	81,350	6,490	8·0

From these figures it will be seen that the second quinquennial period of life is the most common age attacked, then the first, and after that the remaining five-yearly periods in order of succession. Children under a year old are not very prone to take scarlet fever; of 17,310 children under five only 540 were under one. Very rarely infants are born suffering from the disease, and they may become affected within a few days or weeks of birth. An attack has also been met with in a person over ninety years of age. Though, doubtless, the immunity of the later years of life is largely due to the protection conferred by a previous attack, nevertheless unprotected adults appear to be much less liable to scarlet fever than children.

As regards *sex*, it is found that more females than males are attacked at all ages.

The *fatality*, or *case mortality*, is very greatly influenced by age. Generally speaking, the younger the patients the higher the fatality. This is well shown by the statistics of the Hospitals of the Metropolitan Asylums Board. Of 17,310 patients under five years of age admitted during

the years 1888 to 1894, the case mortality per cent. for each year was as follows :—

Under 1	...	29·6
1 to 2	...	26·6
2 „ 3	...	20·7
3 „ 4	...	14·6
4 „ 5	...	10·3,

the total fatality amongst these cases being 16·2 per cent.

From the tables given above it will be seen that the fatality is highest in patients under five years of age, and is especially high amongst infants in the first and second years of life, while it is lowest between the ages of ten and fifteen.

Among the same patients the gross fatality was 1 per cent. higher for males than for females.

The case mortality also varies in different years and epidemics.

The **incubation period** is invariably less than a week. Usually it is three days (seventy-two hours); but it is not infrequently less, and may be of only a few hours' duration.

Clinical History.—Scarlet fever is a disease in which there is much variation in the course and severity of the attack. We shall consequently describe several varieties. It must, however, be understood that these varieties run into one another, and it is often difficult to put an individual case into any definite category.

USUAL FORM OF SCARLET FEVER.

The *onset* is generally sudden, and usually begins with an attack of vomiting, accompanied by chilliness and sore throat; convulsions are decidedly rare. In adults the throat often feels sore for some two or three days before any marked constitutional symptoms set in. In children the vomiting is at times accompanied by transient pallor

and collapse, and it often happens that no complaint is made of sore throat. The temperature quickly rises, reaching in a few hours 101° to 104° Fahr. The face is flushed, and the skin hot and dry. In children the pulse rate is often extremely frequent, reaching 160 per minute without necessarily indicating a severe attack. In adults the pulse generally corresponds with the temperature. The tongue becomes covered with a white fur, and on examining the throat the fauces are found to be reddened and swollen.

The *rash* often appears within a few hours of the commencement of the attack, and in moderate cases is not often delayed beyond twenty-four hours. Exceptionally it commences as a uniform blush, which assumes in a few hours the characteristic punctate appearance to be immediately described ; but usually it is punctate from the very beginning. It comes out first on the upper part of the chest and the neck, and quickly spreads to the rest of the trunk and the upper and lower extremities ; but frequently it makes its appearance over all these parts at once. It may take any time from a few hours to three or four days, or even longer, to attain its maximum development. It is best seen on the chest and neck, in the axillæ, bends of the elbows, flanks, groins, and inner and upper portions of the thighs. It consists of minute, bright red points, situated on a less brilliantly red background, though sometimes there is normal skin between the points. The rash disappears on pressure, except when it becomes petechial, as it often does on the neck, in the axillæ and bends of the elbows, and especially in the creases of the skin. The hæmorrhages are minute and closely set. The eruption cannot be felt, except in certain cases round the hair follicles.

There are certain portions of the skin that are almost

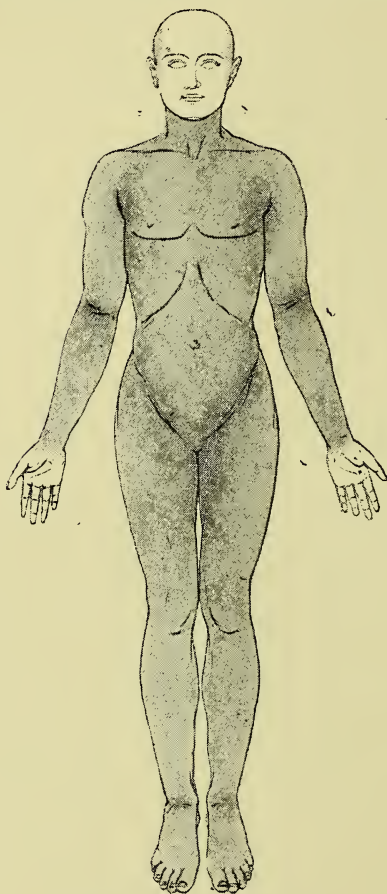


DIAGRAM I.—SCARLET FEVER.*

A uniform, punctate erythema. The face, scalp, palms, and soles are unaffected. The rash is often especially well marked in the flexures of joints.

always unaffected by the rash—namely, the face and scalp, the palms and soles. The rash often extends up the neck

* These and subsequent figures are diagrammatic. They represent the distribution of the rash in typical cases.

in front of the ears to the temples ; but the cheeks, though deeply flushed, do not show the punctate appearance.

A characteristic sign is the "circum-oral ring," which is

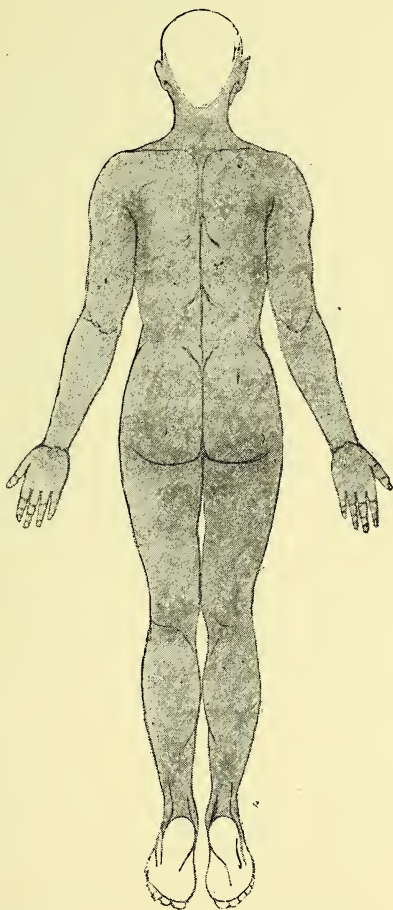


DIAGRAM II.—SCARLET FEVER.

usually well marked in children, though in many cases, especially in adults, it is absent. The skin around the mouth and nostrils is bloodless and pale, affording a

striking contrast to the flushed cheeks. This circum-oral ring is not, however, entirely the result of contrast; there



DIAGRAM III.—SCARLET FEVER.

In some cases of scarlet fever the rash is blotchy on the extremities, while the usual punctate erythema is to be observed on the trunk. In these cases the palms and soles may be affected.

is an actual constriction of the cutaneous arterioles in this region, for the ring can often be most distinctly seen

when the cheeks are not flushed, or may persist for some time after the flush has disappeared.

The rash is frequently accompanied by much irritation and consequent scratching of the skin. When the rash is profuse the skin may be swollen. Miliaria are by no means uncommonly present, showing as little white vesicles with turbid contents.

There are certain varieties of the rash to be described. It may be very slight with respect to its intensity, and then the punctate character is not well marked; it may be limited in extent, and confined to the chest or abdomen; again it may be entirely absent, especially in very mild cases of the disease, and that more frequently than is generally supposed. It may vary in tint from a light pink to a dark red colour. On the extremities, and especially on the wrists, ankles, and the dorsa of the hands and feet, the uniform punctate redness is not infrequently replaced by papules and maculæ, as in measles. In such cases the palms and soles may also exhibit the rash, and in rare instances it is papular upon the trunk also, but even then the face is unaffected. On the front of the legs in adults the redness is often limited to small areas around some of the hair follicles, these areas being separated by much larger areas of normal skin.

The duration of the rash is from twenty-four hours to nine or ten days. It lingers in places where it has been most intense, and petechiæ will often remain for some time after the rest of the eruption has disappeared.

The aspect of the *throat* varies much, even in moderate cases. The soft palate, tonsils, and pharynx may be :—(i) normal or slightly reddened; (ii) reddened and swollen; (iii) swollen, with an exudation of tenacious

mucus upon the tonsils. Occasionally some of the tonsillar follicles are at the commencement plugged with exudation, so that there is somewhat the appearance of a follicular tonsillitis. This condition is not, however, common.

The submaxillary and cervical *glands* are very often swollen and tender. The *tongue* is at first covered with a white fur, through which the swollen filiform papillæ project as red points; but a desquamation quickly occurs, and the fur begins to clear off, leaving the surface red and raw. This desquamation does not take place all over the tongue at once, but in patches. The result is that the surface of the tongue shows patches and often longitudinal streaks of white fur, separated by islets or strips of red. After a time the whole of the fur is completely shed, and the enlarged papillæ standing out prominently upon the red, raw dorsum give rise to the condition known as the "strawberry tongue." Although this condition is usually seen later in the course of the disease, yet it is not uncommon to find it at a fairly early stage, while the rash is still out. The *bowels* are sometimes loose; but as a rule there is constipation, and a purge is required. *Thirst* is often a prominent symptom. The *urine* is of the usual febrile character, being high coloured, and depositing urates. A trace of albumen is often present, especially when the temperature is high. The rate of *respiration* is increased in correspondence with the temperature. The patient is restless, and there is sometimes slight delirium at night.

With the progressive outcome of the rash the *temperature* generally continues to rise one or two degrees, unless it has been very high from the beginning. With the disappearance of the rash the temperature slowly falls, and reaches

the normal at about the seventh to the tenth day of the illness; a fall by crisis is occasionally, but not commonly, seen. With the fall of temperature all the other symptoms subside, and the morbid condition of the fauces disappears.

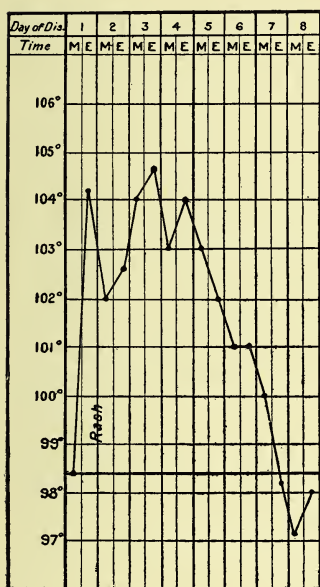


CHART A.

Male, aged 15. Sharp, but uncomplicated attack of scarlet fever, showing the usual defervescence by lysis. Rash early on second day, and lasting till seventh.

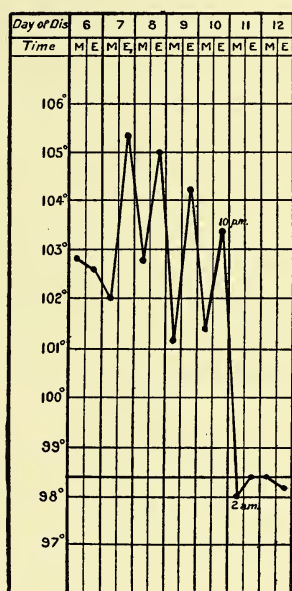


CHART B.

Female, aged 9. Chart of an uncomplicated case of scarlet fever, showing an unusual defervescence by crisis within four hours.

For the first week or two of convalescence the temperature and the pulse-rate may be sub-normal.

With the subsidence of the febrile symptoms a characteristic *desquamation* sets in. This may indeed begin while the rash is still out; on the other hand, its commencement

may be delayed for some weeks, but as a rule it begins towards the end of the first week. The more profuse the rash and the more pronounced the fever, the more marked is the desquamation. It begins first on the upper part of the chest, and on the neck, face, and ears. In the most common form of desquamation small white points appear, looking not unlike miliaria, but consisting of slight elevations of the horny layer of the epidermis. The breaking or rubbing off of the tops of these gives rise to little rings; these rings increase in size, and by their coalescence with adjoining rings irregular gyrate or serpentine figures are formed. Soon the rings and figures are lost in a universal flaky desquamation. In a second form desquamation occurs simply as a branny shedding of the superficial layer of the epidermis. While in a third form large flakes are detached, and the epidermis of the hands and feet comes off like a glove.

It is rare for the skin to be left raw by the desquamation; and it is also rare for the nails to be shed, though the latter may be marked by a transverse groove. There is usually some, and not infrequently much, loss of hair.

A second desquamation over some portions of the skin is not uncommon, while a third and even a fourth have been observed; these are often confined to the palms and soles.

MILD FORMS.

There is a mild form of the disease in which the constitutional symptoms are slight, the principal being sore throat and moderate fever, though in a few cases the temperature may not be raised; the fauces are reddened, but there is little swelling; the rash is either

faint, and only to be observed when the patient is warm in bed or after a bath, or it is absent altogether. Even when present it may be limited to the upper part of the chest. The nature of the disease is often recognised only by the subsequent desquamation, or by the occurrence of some characteristic complication, or by the spread of infection to other persons. The interest and importance of these cases lies in the diagnosis; for the symptoms are often so slight that no complaint is made, and even a careful inquiry will often fail to elicit the occurrence of malaise.

Another mild form of scarlet fever is that in which the eruption is well marked, but constitutional symptoms are almost entirely wanting; even the temperature may not rise one degree above the normal.

SCARLATINA MALIGNA.

In the most severe or malignant forms of scarlet fever the patients are profoundly affected by the poison of the disease, and a very large proportion of those attacked succumb. The symptoms are often severe from the outset, but occasionally they become serious a day or two after the commencement of the attack.

In the worst form the patient is seized with high fever, delirium, vomiting, and collapse, and dies in a few hours, before the rash has developed. Such cases are fortunately very rare, and can only be recognised when they give rise to other instances of the disease.

In the usual form the chief symptom is cardiac failure. The heart beats with great rapidity, and its first sound is short and faint, while the pulse is very weak, and may be irregular. The skin, where not covered by the

rash, is of a yellowish waxy hue, and there is often cyanosis. The respirations are rapid, shallow, and of a sighing character. The temperature is usually high (104° to 106°); but it may never rise above 102° . Vomiting and diarrhœa, especially the former, are often prominent

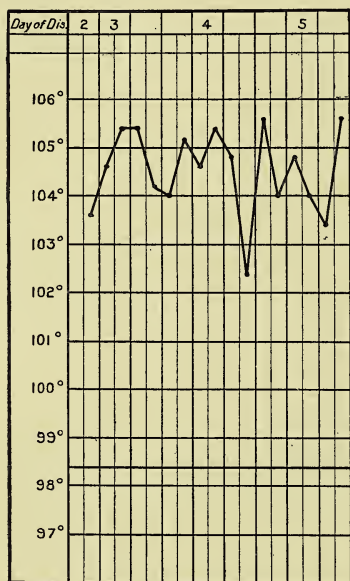


CHART C.

Female, aged 23. Four-hourly chart of an uncomplicated case of malignant scarlet fever, fatal on the evening of the fifth day. On that day the pulse was 192 and respiration 38 per minute.

symptoms. There is generally much restlessness, the patient continually tossing about in bed. Delirium may be, but is not necessarily, present. The fauces are swollen and œdematous, and there is much sticky adherent mucus present, rendering a good view of the parts difficult to obtain. The rash is often profuse; occasionally it is scanty. Its appearance also may be delayed. Death usually occurs within a week; rarely does recovery take place.

A rapidly fatal *hæmorrhagic form* of scarlet fever has been described by certain writers. Hæmorrhages take place into and

beneath the skin and from the mucous membranes. It is probable that some of the cases described under this head were cases of hæmorrhagic small-pox. At any rate, this form of the disease is exceedingly rare, and the authors have no acquaintance with it.

SCARLATINA ANGINOSA.

In this variety a septic condition of the fauces is the most prominent feature of the attack. Usually the symptoms, including those referable to the affection of the fauces, are severe from the commencement ; but sometimes the throat symptoms develop a few days after the disease has manifested itself, and are, as it were, superadded to what was in the first instance an ordinary attack of scarlet fever. The throat symptoms begin with much α -dema of the tonsils and soft palate, which are also covered with mucus, sometimes loose, sometimes very sticky and tenacious ; or a more definite pultaceous exudation may be observed, and in a few cases even membrane, so that the appearance closely resembles and may be mistaken for diphtheria. An ichorous discharge runs from the nostrils excoriating their margins. The swelling of the parts about the pharynx may be so extreme as to obstruct the respiration. It is often difficult, on account of the viscid mucus that adheres to the affected parts, to say exactly what is the condition of the mucous membrane beneath. These cases often prove fatal within a week or ten days ; but should the patient survive for that length of time it will be found that either ulceration or sloughing has taken place.

Ulceration is found in those cases in which the swelling has not been very extreme. It especially affects the angles between the uvula and the arches of the palate, but it is often observed along the edge of these latter parts, and may extend over the whole of the soft palate. The ulcerated portions have a dirty-whitish appearance, simulating membrane. Occasionally the ulceration can be made out to spread slowly from day to day.

When there has been much swelling, necrosis *en masse* of some or all of the parts may be seen. The whole of a tonsil may be involved in the gangrenous process, or the uvula, or one arch of the palate. In rare cases the whole of the soft palate may slough away. It is in cases of this description that fatal hæmorrhage from one of the blood-vessels may occur. In a few cases localised gangrene takes place. When the fauces become free from mucus one or more greenish-white patches can be seen on the anterior pillars of the palate, towards their junction with the mucous membrane of the side of the mouth. In a day or two these patches are found to be replaced by holes, the size of which varies from that of a large pin's head to that of a sixpenny piece. If the perforation is a large one it is usually oval, its long diameter being parallel to the free edge of the anterior pillar. These perforations have very clean cut edges. Sometimes the remaining portion of the anterior pillar breaks away, and a large gap is left. When recovery takes place there is much shrinking, so that after some weeks it may be difficult to say where the perforation has existed, it having been reduced to a mere pin-point. On the other hand, large perforations only partially contract, and a hole, of which the size varies in different cases, permanently remains.

In all the forms of scarlatina anginosa stomatitis more or less severe is often seen. Aphthous patches or superficial ulcers are observed on the tongue and buccal mucous membrane, and the lips and angles of the mouth become excoriated. The breath is offensive, at times highly so.

In most cases the cervical glands are much enlarged, forming very obvious swellings on each side of the neck; and suppuration nearly always results. The cellular tissue of the neck is liable to be the seat of inflammation,

and the skin becomes tense, hard, and brawny. In the most extreme form of this complication the whole space between the jaw and the clavicles and sternum becomes affected, and the condition is then known as "bull-neck." Blebs form on the skin, which becomes of a bluish hue; then the skin gives way in one or more spots, and a thin, offensive, slightly purulent fluid escapes. If a probe be introduced into the hole the skin will be found to be undermined to a greater or less extent, and when it is slit up the subcutaneous tissue will be observed to have sloughed. On the removal of the slough the muscles, nerves, and other structures will be seen, as it were, dissected out.

The rash is usually marked, but now and then it is slight. It may be very blotchy on the extremities, particularly about the elbows, wrists, and knees. In prolonged cases, the secondary rashes, to be described later, are not infrequently seen.

Together with the local changes in the fauces there are marked febrile symptoms; the temperature runs up to 105° or 106° , and is often 103° for days together. The pulse is very frequent; at first it is full and bounding, but it becomes gradually smaller and more feeble. The respirations also are increased in frequency, and in very severe cases may become of the Cheyne-Stokes variety. The patient is restless and delirious; and when death occurs it is frequently preceded by coma. The aspect of the patient is that of one suffering from septicæmia. In all these cases, fatal or otherwise, there is rapid and marked emaciation. Death may occur during the febrile stage before the end of the first week with symptoms of prostration (feeble pulse, lividity, etc.), or during the second week from exhaustion. In other instances the

temperature regains the normal, and to a certain extent the condition of the patient improves; but the improve-

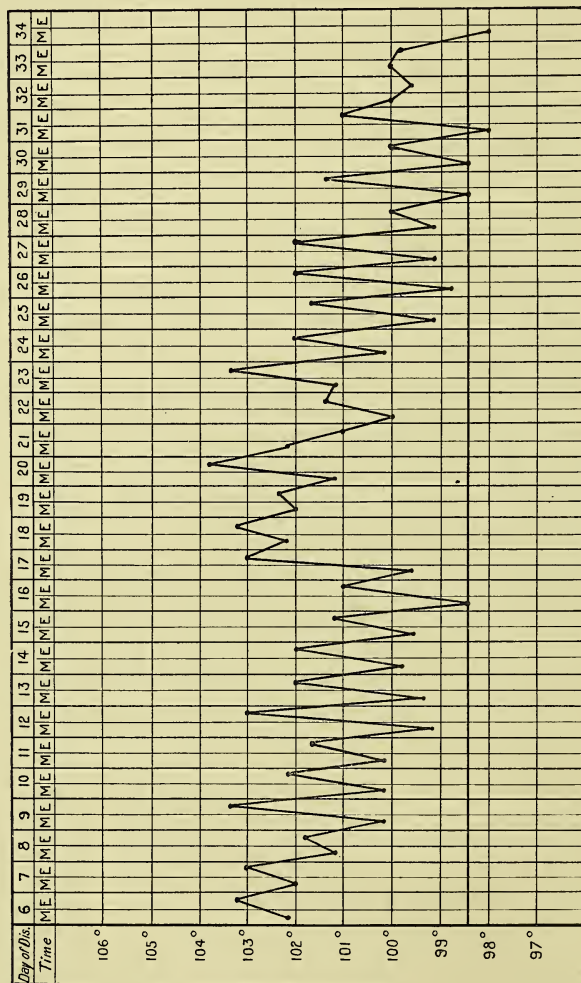


CHART D.

Male, aged 13. Typhoid form of scarlet fever; uncomplicated, and ending in recovery.

ment is only temporary, and the patient after all sinks from exhaustion, the fatal termination being heralded by persistent diarrhoea and vomiting.

Cases of anginous scarlet fever are very frequently complicated by otitis, glandular abscesses, and other septic conditions. The mortality is high; and when recovery takes place convalescence is slow and protracted.

TYPHOID FORM OF SCARLET FEVER.

Here the throat symptoms may be slight and the rash normal, but the fever is high from the beginning; and instead of the febrile symptoms subsiding at the end of seven or eight days they continue for some three or four weeks, and the temperature is of a remittent type. The mucous membrane of the fauces often becomes granular, and perhaps a little ulcerated. The cervical glands are enlarged. There is marked emaciation, otherwise nothing abnormal can be made out, except in some cases an enlargement of the spleen. A secondary roseolous eruption over the whole body is not uncommon about the end of the third week. Where the case ends fatally death is usually by exhaustion. Post-mortem it is sometimes found, as in other forms of the disease, that the mesenteric glands and the lymphoid tissue of the intestines, especially the Peyer's patches, are swollen. Ulceration of the intestines does not occur. There is, in fact, no evidence to show that the cases in question are instances of enteric and scarlet fever occurring in the same patient at the same time. The typhoid form is probably a septic variety of the disease.

PUERPERAL SCARLET FEVER.

The relationship between scarlet and puerperal fever has been the subject of much discussion. Some authors believe that the scarlatinal poison may give rise in puerperal women to septicæmia unaccompanied by signs of scarlet fever.

Others consider that there is no connection between the two diseases, and that the poison of scarlet fever produces in puerperal women an ordinary attack of scarlet fever, and never a puerperal septicæmia. One of the difficulties that has given rise to much confusion is the fact that a rash may occur in puerperal fever, as in other forms of septicæmia, and that this rash may be mistaken for the rash of scarlet fever. Such cases are put down as cases of puerperal fever caused by scarlet fever, and the rash is taken to be an evidence of the infection of scarlet fever. Again, it is quite certain that women shortly after confinement may contract scarlet fever, and that the attack may run a course that is quite usual, and not in any way influenced by the puerperal state. Many such cases have been recorded; as an instance we will quote the following. A lady was confined normally; within four days she was attacked with scarlet fever, which ran a mild course followed by desquamation, without complications, and without any sign of pelvic trouble. That the case was one of scarlet fever was shown by the fact that the infant contracted the disease five days after the mother was first affected. Both mother and child were admitted into the London Fever Hospital.

It is also quite certain that women, both immediately before and after confinement, have often been exposed to the infection of scarlet fever, without, however, an attack either of scarlet fever or of puerperal fever having followed. On the other hand, in many instances of puerperal septicæmia the only source of infection traceable has been in connection with cases of scarlet fever.

The view we take of the subject is as follows. The virus of scarlet fever "breeds true" whether it attacks individuals in the possession of perfect health, or women

in the pregnant or puerperal state. Nevertheless, many cases of scarlet fever are complicated with septic troubles, such as sloughing of the fauces, glandular and other abscesses, and even pyæmia; and cases complicated by affections of this nature may give rise to puerperal septicæmia, should proper antiseptic precautions not be carried out, just in the same way as puerperal fever may be caused by any other septic disease. Whether under these circumstances the attack of puerperal fever is accompanied by scarlet fever depends, to a large extent, upon whether the patient is protected or not by a previous attack of scarlet fever. There is no strong evidence to show that pregnant or puerperal women are more or less liable to contract scarlet fever than are other women, nor that when attacked they are more severely affected.

SURGICAL SCARLET FEVER.

There is no evidence that scarlet fever occurring in patients the subjects of operation or other wounds differs in any respect from ordinary scarlet fever, either in its course or in its severity; nor does it appear that such patients are more liable to contract the disease than other patients.

We must consider rather more fully the occurrence of scarlet fever amongst patients suffering from *burns*. Two questions arise in this connection. Firstly, are patients, the subjects of burns, more liable than other surgical patients to be attacked with scarlet fever? Secondly, are scarlatiniform rashes occurring in those suffering from burns of a septic nature, or are they evidence of scarlet fever? With regard to the first question we are indebted to Mr. G. B. Smith, Surgical Registrar to Guy's Hospital, for the following facts. During the years 1891, 1892, and 1893, there were

admitted into that Hospital 224 cases of burns or scalds, and of these 5 (2·2 per cent.) developed scarlet fever. During the same period 10,620 patients suffering from other surgical diseases were admitted, and of these 14 (·12 per cent.) developed scarlet fever. It must be remembered that the majority of the patients suffering from burns were children, while the majority of the other surgical patients were adults, and that children are much more liable to be attacked with scarlet fever than adults. But, as a matter of fact, about a quarter of these 10,620 patients were children. So that even when the question of age is taken into consideration it would appear that patients suffering from burns are especially liable to be attacked with scarlet fever. Again, during the year 1893, there were admitted into the Eastern Hospital 29 surgical patients suffering from scarlet fever, and of these 6, that is about one-fifth, were cases of burns; a large proportion, seeing that the proportion of burns to other surgical cases in a general hospital, as obtained from the statistics of Guy's Hospital given above, is about 1 to 47. In answer to the second question we can only state that in our experience the cases of burns admitted into a fever hospital as cases of scarlet fever are really such. Otherwise many of them would subsequently develop scarlet fever, an occurrence decidedly uncommon. Moreover, they present the other symptoms characteristic of that disease.

Complications.—Complications may arise at any stage of the disease, from its commencement until desquamation has been completed. Some complications, however, occur more frequently during the febrile stage, others during convalescence, while others show no preference for any particular stage. For example, otitis may occur at any

time; nephritis usually starts during convalescence, but may begin during the first few days of the attack; arthritis is most common during the period of defervescence, but may occur earlier or later.

It will be convenient to divide the complications into two groups, the early and the late; but it must be understood that no very sharp line can be drawn between them.

Early Complications.—Those occurring during the febrile stage.

Throat.—Gangrene and ulceration, with the corresponding glandular affections, have already been described under scarlatina anginosa. Suppurative tonsillitis may occur, but is rare.

A *membranous condition* of the fauces has already been mentioned as occurring during the febrile stage in certain cases of scarlatina anginosa. This is seldom true diphtheria, as is shown by the fact that it is rarely followed by paralysis, and that the bacillus diphtheriæ is not often present. Occasionally, however, true diphtheria may complicate the acute stage of scarlet fever. The diphtheria of convalescence will be described later.

Rhinitis is frequent, especially if the throat affection is severe; it may, however, occur when the sore throat is slight. The condition shows itself by an acrid discharge from the nose, causing excoriation of the nostrils. Later, the discharge becomes mucopurulent.

Conjunctivitis and *blepharitis* are not uncommon, especially among children. The former may be followed by ulceration of the cornea.

Dilatation of the heart may occur apart from nephritis. The heart's action becomes irregular; the apex is displaced outwards; the first sound is shortened, and is often replaced by a murmur. As the patient recovers the apex

returns to its usual position, the action becomes regular and the sounds normal.

Pericarditis sometimes occurs, but is not common, while *endocarditis* is decidedly rare.

Bronchitis is not uncommon amongst children ; it may be combined with *lobular pneumonia*.

Pleurisy, *empyema*, and *lobar pneumonia* are rare.

Laryngitis and *ulceration of the larynx* are met with in some cases of scarlatina anginosa, or when there is extensive cervical cellulitis. They may necessitate tracheotomy.

Albuminuria.—When the temperature is high the urine may contain a little albumen, which disappears as the fever subsides ("initial albuminuria"). An early nephritis occasionally occurs, and must be distinguished from this "initial" or "febrile albuminuria."

Arthritis or *Rheumatism* usually comes on as the temperature is subsiding ; that is about the end of the first week. Of 3,026 cases of scarlet fever admitted to the South Eastern Hospital and analysed by Dr. Hodges, 117 were complicated by rheumatism, or 3·8 per cent. It occurs most frequently between the ages of eight and twenty-five. The joints most constantly affected are those of the hands and the wrists, but the knees, elbows, and other joints may be involved. They become painful, and there is sometimes effusion and redness. There is generally moderate pyrexia. The arthritis usually lasts less than a week. The relation of this arthritis to ordinary acute rheumatism is interesting ; it resembles the latter in the fact that it readily yields to treatment with salicylate of soda, and that it flies from one joint to another. It however differs from acute rheumatism in the absence of profuse perspiration, and in the rarity of occurrence of endo- or pericarditis. It also does not appear to

recur so often as ordinary acute rheumatism. Dr. Hodges noted a return of pain in the joints in 13 per cent. of his cases.

We are inclined to believe that scarlatinal rheumatism has no etiological connection with acute rheumatism. Nevertheless, it must be remembered that patients who have previously suffered from acute rheumatism are prone to a recurrence of it when attacked by scarlet fever.

In some cases of scarlet fever *suppurative arthritis* occurs; but such cases are of a pyæmic nature, and probably are not connected with the ordinary scarlatinal rheumatism.

Otitis media may come on at any time during the disease. It is more common in cases where the throat affection has been severe, but often occurs when this has been mild. Sometimes it is ushered in by severe pain in the ear, accompanied by a rise of temperature; but often pain is absent, and a discharge from the ear is the first sign of anything being amiss. Frequently both ears are affected. As a rule the inflammation leads to the formation of pus, and to perforation of the tympanic membrane. The pain, when present, is not of long duration, and under treatment the discharge ceases and the perforation, unless large, closes. The hearing is not much affected; occasionally, however, the patient becomes quite deaf for a time. In such cases much improvement usually ensues, and it is now quite uncommon for patients to leave the hospital with much impairment of hearing.

Otitis may lead to mastoid disease, meningitis, thrombosis of the lateral sinus, cerebral abscess, or pyæmia; but these complications are rare if treatment is efficiently carried out.

Otorrhœa may also arise from inflammation of the external auditory meatus, the middle ear being unaffected.

Cervical cellulitis has already been mentioned as complicating anginous cases; but it may occur in cases where the throat symptoms are slight.

Late Complications.—After the fever has subsided and the patient is convalescent, several complications are likely to set in. The most usual time is at the end of the third week, but their occurrence may be postponed as late as the sixth or seventh. The most common are nephritis and adenitis.

Nephritis usually occurs about the end of the third week; it may begin quite early, during the first week, or in rare instances as late as the twelfth. It does not appear to bear any relation to the initial albuminuria already mentioned.

The nephritis begins in various ways. Sometimes the patient does not appear to be constitutionally affected, and the renal mischief is first revealed by an examination of the urine. Occasionally the albuminuria is preceded by a rise of temperature a day or two beforehand. Usually, however, the patient feels languid, and complains of headache, loss of appetite, and sometimes pain in the back. Vomiting is by no means uncommon. The temperature is raised, and may reach 103° or 104° . Other complications, especially adenitis, are apt to manifest themselves at the same time. In the urine there may be at first only a trace of albumen, which gradually increases in quantity and then gradually disappears. Blood is usually present, even when the amount of albumen is small. Sometimes a large amount of albumen with much blood, granular *débris*, and casts (epithelial and blood), appears at the very onset of the nephritis. At other times a slight, may pass on to a severe, albuminuria with blood and casts. The quantity of albumen may

vary considerably from day to day ; it may even disappear, to return after a day or two, especially when the nephritis is clearing up.

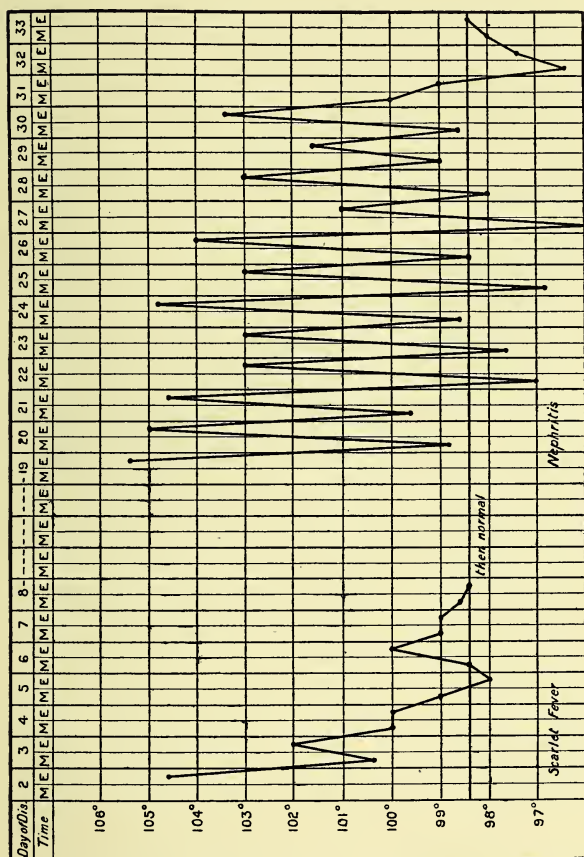


CHART E.

Mild attack of scarlet fever ; acute nephritis on nineteenth day. The chart is an extremely well-marked example of the "spiking" observed in some cases of nephritis. The patient was a girl, aged 5. There was no other complication.

Very often there is little in the way of constitutional symptoms. In other cases the temperature keeps high, and often shows marked remissions, giving the tracing on the chart a spiked character, as in pyæmia. Headache

and vomiting continue, the patient becomes very anæmic, and rapidly loses flesh ; the pallor and wasting are often extreme. There may be a little puffiness about the eyelids and face, but extensive œdema is uncommon in patients who have been treated from the commencement of the attack. The cases in which dropsy supervenes are usually those in which the primary attack of scarlet fever has not been recognised on account of its mildness, or has been neglected. In the former group of cases the first intimation of anything wrong may be the occurrence of dropsy.

In most cases of nephritis the amount of urine passed is diminished, and often a deposit of lithates is observed. As recovery takes place the amount of urine increases, and a larger quantity than normal may be voided. In severe cases the diminution during the acute stage of the nephritis may be extreme, and absolute suppression may occur.

When blood is present the urine may be of a bright red hue, but is more often of the brownish tint known as "smoky," depositing a reddish-brown sediment. During recovery the colour is a pale yellow, and the urine is quite clear.

Drowsiness is not uncommon ; in severe cases there are convulsions and coma. In some patients the arterial tension is raised, an event of by no means universal occurrence ; on the contrary, in many cases the pulse is soft and compressible.

The nephritis may be complicated by other affections, of which the most common are inflammation of the serous membranes, especially the pleura and pericardium, lobar pneumonia, and acute dilatation of the heart. The latter is a not infrequent cause of sudden death. Secondary

rashes (to be described later) are at times met with. Neuro-retinitis practically never occurs.

It is very common for nephritis to be accompanied by some other complication of scarlet fever, such as adenitis, secondary tonsillitis, or otitis.

The duration of the inflammation of the kidneys is very variable. Commonly it is about four weeks; it may be more or less. Sometimes the acute passes into a chronic nephritis.

The frequency with which nephritis occurs seems also to be very variable. In some epidemics and in some years it is seen more often than in others; the extremes were 4 and 17 per cent. in a series of years for patients admitted into the London Fever Hospital; in the year 1893 the incidence was 13 per cent. in the hospitals of the Metropolitan Asylums Board (Dr. Meadows Turner). Though no age is free from the risk of nephritis, it occurs with greatest frequency during the second quinquennium. The case mortality in the series of cases at the London Fever Hospital was nearly 7 per cent., in Dr. Turner's cases 8·1 per cent.; it is highest under five years of age.

There is no relation between the character of the attack of scarlet fever and the occurrence or severity of a subsequent nephritis.

Dropsy without Albuminuria has been described by certain writers as a complication of the convalescent stage of scarlet fever; it is, however, exceedingly rare.

Pyæmia is fortunately an uncommon complication. It occurs generally in cases of severe throat affection or of middle ear disease. The symptoms are the usual ones—a high temperature, rigors, suppuration of joints, and formation of abscesses in various places, often in

connection with the periosteum. The joint affection may in its early stage be mistaken for rheumatism. Recovery may ensue.

Late *Adenitis* occurs at about the same time as, and is often accompanied by, nephritis. The submaxillary or the cervical glands become swollen and tender, and there is generally a rise of temperature, even to 103° or 104° . As a rule, the temperature falls in a few days, and the swelling of the glands subsides, but suppuration may result. The amount of febrile disturbance does not always coincide with the amount of glandular affection. In fact, at this period there may be pyrexia, lasting for a day or two without any obvious complication.

Secondary Throat Affections.—During convalescence the tonsils may again become inflamed, causing either a *simple* or a *follicular tonsillitis*.

True *Diphtheria* is not uncommon, and may arise at any stage of the disease. The most common period for its onset is during convalescence, after the primary symptoms of scarlet fever have subsided. But it must not be forgotten that diphtheria may complicate scarlet fever at the very commencement of the attack, and may easily be mistaken for the angina of the latter disease. When the diphtherial affection is mild, there may be nothing in the appearance of the throat to suggest diphtheria. A bacteriological examination is the only means of arriving at a diagnosis. The same difficulty may arise in diagnosing diphtheria occurring during the convalescent period, and every case of sore throat at this stage should be looked upon with suspicion; so too should rhinorrhœa, for the nose may be the part primarily or solely affected.

As far as the clinical aspect of post-scarlatinal diphtheria

is concerned, it does not differ from a primary attack of diphtheria. It is stated, however, to be more fatal; but this may be partially due to the fact that the mild cases have passed unrecognised.

There has been much discussion as to the causation of this complication. So far most of our information on the subject has been obtained from the experience of fever hospitals in which large numbers of patients have been aggregated. Among many assigned causes are overcrowding and defective drainage; and no doubt these conditions will favour the development of the disease. An important factor in its causation is the introduction of an unrecognised case among the scarlet fever patients; and when once this has happened the disease will spread in the usual manner, for particulars of which we refer the reader to the chapter on Diphtheria.

In fever hospitals, unless proper precautions are taken, diphtheria may be conveyed from the diphtheria wards by the attendants or by means of fomites.

Stomatitis is by no means infrequent in children, especially if their surroundings are hygienically unfavourable. The gums become spongy and bleed readily, and superficial ulceration occurs. Aphthous patches form on the tongue and buccal mucous membrane. In rare cases a gangrenous condition allied to noma follows.

Eczema, of an impetiginous nature, on the face and head is common, especially amongst the children of the lower classes. It most commonly affects the skin about the ears and nostrils, and is very often the result of irritation caused by chronic discharges from those organs.

Vaginitis is also of common occurrence.

Secondary rashes.—By “secondary rash” we mean one that makes its appearance after the primary and character-

istic rash of the disease has disappeared, excluding cases of relapse. Secondary rashes are not very uncommon; they are most frequently seen during the second and third weeks, occasionally later.

By far the most common form of the secondary rash is a papular eruption which appears on the extremities. The papules, which are of a light rose-red colour, are seen first on or about the extensor surface of the elbows and knees, and on the buttocks; during the next day or two fresh papules come out on the remaining parts of the limbs, more, however, upon the extensor than upon the flexor surfaces. The papules are at first discrete, but after a time they run together to form irregular maculæ and blotches; in fact the rash may be macular from its commencement. In some instances the papules are very firm to the touch; and flat, elevated, red patches may be seen, as in lichen planus. Exceptionally the trunk and face are affected, in which case the eruption is very similar to that of measles. The duration of the rash is from one day to a week, or even more.

Less common forms of secondary rashes are a bright scarlatiniform erythema, on the trunk and extremities, and an urticarial eruption affecting chiefly the limbs. The former is like the primary rash, but there is no sore throat, little, if any, rise of temperature, and no other symptom of an attack of scarlet fever, as there would be in a relapse.

Secondary rashes are commonly met with in cases of scarlatina anginosa; in such cases there may be some fresh adenitis accompanying the rash. Again, these rashes not infrequently accompany nephritis, at any stage of which they may appear. Lastly, very occasionally, a secondary rash may occur during convalescence from

a mild uncomplicated attack of scarlet fever, and its appearance may be unattended with any constitutional disturbance. The occurrence of these rashes in no way influences prognosis.

Amongst *rare complications* of scarlet fever may be mentioned *jaundice, meningitis, peritonitis, and mania* (post febrile).

It is to be observed of all complications that they may be met with either singly or in combination.

Relapses.—By relapses we mean second attacks of the disease occurring before the desquamation of the primary attack has finished. In the relapse the fever, rash, and sore throat are present, and the usual desquamation follows. As an example we will quote the following :—A boy, aged six years, was admitted into the Eastern Hospital, on January 28th, with typical scarlet fever, which was followed by desquamation. On February 20th he was attacked with fever and sore throat, and on the next day the characteristic punctate eruption appeared. A second desquamation followed.

Usually relapses are mild ; but they may be severe, and even lead to a fatal termination. They are uncommon.

Second attacks and protection.—Although one attack of scarlet fever undoubtedly affords protection against subsequent attacks, nevertheless second attacks are not very uncommon, and third, and even fourth attacks may occur.

Period of Infectivity.—The infectivity begins at the earliest stage of an attack, but is probably greatest when the fever is at its highest. Its duration varies in different cases, but in most instances the patient is free from infection at the end of six weeks. Occasionally, however, a patient who has to all appearance completely recovered, who is the subject of no complication, and who has quite finished

peeling, remains infectious for a much longer period, as is shown by his giving rise to fresh cases of the disease. It is impossible to say, with our present ignorance of the cause of scarlet fever, in what part of the body the infection lingers; perhaps in the nasal, buccal, or pharyngeal mucous membrane, perhaps in the skin. There is some reason to think that it may persist in chronic discharges from the mucous membranes. No patient should be considered free from infection before the end of six weeks; and it is advisable, though it has not been proved to be necessary, that isolation should be continued till the first desquamation is complete, and while any chronic discharge is present.

Morbid anatomy.—In cases of *malignant scarlet fever*, which is the type of the uncomplicated disease, but little is found *post-mortem*. Even the fauces often show very little change to the naked eye, at most œdema or superficial ulceration; microscopically there is evidence of inflammation. The skin is often livid and discoloured, but the rash has disappeared. Microscopically there is an infiltration of the rete mucosum with leucocytes. The mesenteric glands are not infrequently enlarged, and sometimes the lymphoid tissue of the intestines is also swollen, especially the Peyer's patches. Exceptionally there is enlargement of the spleen. In cases where death has taken place during the febrile period the appearance of the kidneys to the naked eye is normal; but slight microscopical changes of an inflammatory nature have been described as occurring even at this stage. It is exceedingly doubtful whether such changes are in any way connected with those seen in cases of the nephritis of convalescence.

In *scarlatina anginosa* ulceration or sloughing may be observed about the fauces; occasionally one or more

perforations are found in the soft palate. Ulceration may spread to, or occur independently in the pharynx and larynx. The cervical glands are swollen, and may be embedded in infiltrated subcutaneous tissue. Suppuration is common. Abscesses may also be found in other parts, and occasionally one or more joints contain pus.

In *scarlatinal rheumatism* nothing is found in the affected joints beyond a little excess of fluid and hyperæmia of the synovial membrane.

In *scarlatinal nephritis* the appearance of the kidneys will depend upon the period at which death has supervened. During the early days of the nephritis the kidneys are usually swollen, full of blood, and larger than normal; the cortex is increased in width, the distinction between the cortex and medulla is difficult, and both these parts of the organ have a coarse mottled aspect. The capsule peels readily. It is not uncommon to find small hæmorrhages beneath the capsule, and into the tissue of the cortex. Microscopically there is proliferation of the cells lining the glomeruli and the covering of the vascular tufts, hyaline thickening of the capsule, and extravasation of blood or fibrin into its interior. The epithelium of the renal tubules is degenerated, and the tubes filled with blood, fibrin, and epithelial *débris*. The interstitial tissues are infiltrated with leucocytes, both around the glomeruli and between the tubules. The vessels are engorged with blood. These microscopical changes are sometimes found to be unevenly distributed, patches of much damaged tissue alternating with patches of apparently healthy structure.

If death has occurred at a later period of the nephritis the kidneys are usually pale, slightly enlarged, and of a coarse appearance on section. Occasionally, however, they are a little smaller than normal, red, and slightly granular.

In these late cases the ordinary changes of tubal and interstitial nephritis are found.

Sometimes when death has occurred while renal symptoms are present, the kidneys exhibit a normal aspect to the naked eye. But in such cases the microscope always reveals inflammatory changes.

Acute dilatation of the heart is sometimes found in death from nephritis.

Should any other complications have been present the corresponding lesions will be observed.

Pathology.—Practically nothing is known about the real cause of scarlet fever. It is no doubt produced by a living micro-organism, but whether this is a bacterium or a protozoon is quite uncertain.

Probably the tonsils and neighbouring parts are the seat of infection, although we cannot say whether the materies morbi remains localised to these parts, as in diphtheria, or is diffused throughout the body, as in some forms of septicæmia.

Cultivations made from the throat almost invariably reveal the presence of a streptococcus similar to, if not identical with, the streptococcus pyogenes. A description of this streptococcus will be found in the chapter on Erysipelas; and it will be unnecessary here to enter into a discussion upon the signification of the cultural and other differences which exist between streptococci from various sources. It will suffice to say that the streptococcus found in scarlet fever does not differ essentially from the streptococcus pyogenes.

In *scarlatina anginosa* the tissues of the fauces are invaded by the streptococcus, and the septic character of the symptoms is no doubt due to this cause. In diphtheria, as will be seen later, a secondary invasion

of a like nature frequently occurs. It would appear that the condition of the throat in both these diseases favours the growth of the streptococcus.

In many cases the streptococcus remains localised in the tissues of the fauces, but it frequently gains access to the cervical glands, producing inflammation and suppuration. When it escapes into the connective tissue outside the glands it causes cervical cellulitis. In other cases it is carried by the lymph and blood to other parts of the body, and then sets up metastatic abscesses. In the pus from the middle ear in cases of otitis the streptococcus is generally present, but in association with other bacteria, as would be expected from the connection of the middle ear with the fauces through the Eustachian tube. Most of these associated bacteria are harmless saprophytes, but in some cases other pyogenic bacteria are found.

These considerations show the importance of treating the throat with antiseptic sprays and irrigation.

Nephritis does not appear to be caused in the same way as the secondary complications above mentioned, for its advent is quite independent of the intensity of the septic condition of the throat. It is probably due to the excretion through the kidneys of the toxic bodies formed by the true scarlatinal micro-organism, whatever that may be, and it would thus be analogous to the nerve degeneration that follows diphtheria. In rare cases of nephritis streptococci are found in the kidneys, but they are probably not the cause of the nephritis.

Diagnosis.—The diagnosis of scarlet fever may be quite easy or exceedingly difficult. The very mild cases are those which give rise to the greatest difficulties. The rash may be slight or entirely absent, and the

fauces may only exhibit a slight redness indistinguishable from an ordinary catarrhal sore throat. A history of sudden onset attended with vomiting may assist the observer in arriving at a correct conclusion, and so may the presence of a strawberry tongue, or the evidence of exposure to contagion. It must be admitted that in some cases a diagnosis is impossible at first, and it may be necessary to wait until a late adenitis or nephritis appears, or until desquamation occurs, to clear up the nature of the case.

In the most severe form of scarlet fever, where the patient dies before the rash appears, the diagnosis can only be made by circumstantial evidence of exposure to, or transmission of, the disease.

In the early stage of all forms the diagnosis must be deferred until the rash has appeared, although the history of the onset will often render the nature of the case probable.

In the late stages, where the patient is seen with a late adenitis or with nephritis, a careful inquiry will often elicit the history of a previous sore throat, and perhaps of a rash. In such cases a thorough examination must be made for desquamation; as a rule, the nature of the case may be determined in this way.

With regard to diseases that are liable to be mistaken for scarlet fever, they are—firstly, diseases of the throat, such as *diphtheria*, *catarrhal sore throat*, *tonsillitis*, and *follicular tonsillitis*; and secondly, diseases associated with a rash, *morbilli*, *rubeola*, the initial stage of *variola*, *septic eruptions*, and various forms of *erythema*, including *medicinal rashes*.

Diphtheria.—The diagnosis between this disease and scarlet fever is often attended with much difficulty,

from the fact that the faucial affection in true diphtheria is by no means always membranous, and that, on the other hand, undoubted membranous formation may be found in scarlet fever.

In the presence of the scarlatinal rash there can be no question as to the existence of scarlet fever; the only question is whether the patient is also suffering from diphtheria. In cases of doubt a bacteriological examination will settle the difficulty.

Supposing the patient is seen before any rash has appeared (and in cases such as we are considering the outcome of the rash may be delayed), or supposing the rash has disappeared, and there is only the history of its occurrence to guide us, attention to the following points will help in arriving at a diagnosis. In scarlet fever the febrile symptoms are more pronounced than in diphtheria, so that a high temperature, a very frequent pulse, and delirium are in favour of this disease rather than diphtheria. Membranous sore throat with little or no pyrexia is almost certainly diphtheria. Vomiting is much more constantly a prodromal symptom of scarlet fever than diphtheria. Much œdema of the parts underlying the membrane is in favour of scarlet fever, so also is a very red hue of the fauces.

In cases where the exudation is pultaceous, and not distinctly membranous, scarlet fever must be suspected if the febrile and other symptoms that have just been mentioned are marked. The "strawberry" tongue is seen more often in scarlet fever than in diphtheria, in which disease, as in others also, it is occasionally met with; much more characteristic of scarlet fever is the "peeling" tongue. The occurrence of ulceration (unless it be very superficial) and gangrene of the fauces is

exceptional in diphtheria. The perforations of the soft palate, previously described, are characteristic of scarlet fever, though they are not particularly common.

In all cases any history of exposure to the specific infection of scarlet fever or diphtheria, as the case may be, is important.

Catarrhal Sore-Throat.—This condition, in which the fauces are reddened and swollen, is similar to that met with in the milder forms of scarlet fever, and can only be distinguished by the absence of the rash. There is much difficulty in diagnosing between these cases and mild cases of scarlet fever where the rash is absent or slight. As has already been stated, the diagnosis is at times impossible. The same observations apply to *tonsillitis* and *follicular tonsillitis*; but this latter condition is not common in scarlet fever.

No difficulty is to be met with in distinguishing *granular pharyngitis*, *herpes of the tonsil*, and *quinsy*.

Measles (morbilli), before its characteristic eruption has appeared, is not infrequently mistaken for scarlet fever. This perhaps is because in a few cases of measles, on the second or third day, a diffuse erythema may be seen on the chest or over the whole trunk; but the presence of catarrhal symptoms (coryza, sneezing, and coughing), and the absence of marked sore throat, should put the practitioner on the right path.

During the eruptive stage scarlet fever presents in a few cases a similarity to measles. In scarlet fever the eruption on the extremities may be very like that of measles; still more rarely is it so on the trunk. In these cases the sore throat, the freedom of the face from rash, and the absence of catarrh of the respiratory tract serve to distinguish scarlet fever.

Some cases of *rubeola* are very difficult to diagnose from mild scarlet fever; for though the rash of *rubeola* begins in discrete spots, yet in a short time it may become, by a coalescence of the spots, a uniform punctate erythema, and at this stage a difficulty arises. But in *rubeola* the rash may still be seen to be "spotty" on the lower extremities when it has become coalescent on the upper and on the trunk, and it may also be seen on the face, or there will be a history of its having been there shortly before; also in this disease the temperature quickly falls, and the sore throat is slight. The enlargement of most of the lymph-glands that are within reach of the touch is also characteristic.

For the prodromal rash of *variola* see the chapter on that affection.

Septic rashes (often very much like that of scarlet fever) are to be diagnosed by the presence of some unhealthy wound or other cause of septicæmia, and by the absence of sore throat.

In the various forms of *erythema* the affected areas of skin usually have a distinct margin, whereas in scarlet the rash gradually fades into normal skin.

Of *medicinal rashes*, that produced by belladonna most resembles scarlet fever. In poisoning by this drug the throat is dry, and there are in marked cases pyrexia and delirium; the pupils are widely dilated. The rash is of a bright red colour, but is not generally punctate.

Further information concerning the last three kinds of rash will be found in Chapter IV.

Prognosis.—On reference to the paragraph on Etiology it will be seen that scarlet fever has a much higher fatality in patients under five years of age than in those above that age, and that for the first five years of life

the younger the child the more likely it is to die if attacked.

The hygienic conditions under which the patient lives have probably an important influence on the mortality. Under bad hygienic conditions the anginous form is more common, and the fatality of these cases is high.

The character of the epidemic is also of importance, some epidemics being of a mild others of a severe type. The case-mortality of scarlet fever in London for the years 1892 and 1893 was 4·3 per cent.; but epidemics have been recorded in which the case-mortality has been as high as 30 per cent.

With respect to the different forms of the disease, malignant scarlatina is the most fatal, then comes the anginous, and lastly the typhoid variety. Of the mild cases it is to be observed that though the immediate prognosis is good, yet very serious and indeed fatal complications may subsequently arise. Of these the most important are nephritis, otitis, and diphtheria.

In scarlatina maligna, and in most instances of scarlatina anginosa, death is usually due to heart failure; hence a soft, feeble pulse, indistinct cardiac sounds, cyanosis, sighing or irregular respiration, and a waxy and yellow appearance of the skin are signs of evil import. Death may occur suddenly during the first three or four days of the disease, but usually it supervenes gradually at a later period. It is to be looked for in cases when the temperature has continued high, the pulse rate has been very frequent (160 to 200 per minute), and delirium and restlessness have been present for several days together without intermission. In certain fatal cases of scarlatina maligna, however, the temperature is not high.

Death in the typhoid form of scarlet fever is usually the result of exhaustion.

In every class of case persistent vomiting and diarrhœa, especially the former, are unfavourable signs.

With respect to the opinion that is to be drawn from the local affection of the fauces there are two conditions which are especially unfavourable. The first is the presence of much œdema, the second the existence of much exudation. In the former class of case the symptoms of cardiac failure are liable to supervene; but if they do not, and the patient survives the first four or five days of the illness, the œdema is likely to be followed by sloughing with its accompanying risks of septicæmia, or in a few cases by hæmorrhage from the vessels in the neighbourhood of the tonsils. When there is much exudation it is difficult to ascertain the condition of the underlying mucous membrane, but when this has cleared off, ulceration, and sometimes perforation, of the soft palate can be seen. Many cases of this kind recover, though slowly; but there are some in which certain symptoms are met with (bearing no relation to the amount of ulceration), which result in a fatal termination by exhaustion. These symptoms are vomiting, diarrhœa, progressive emaciation (often in spite of the retention of ample nourishment), and a persistently though moderately frequent pulse. These cases are often deceptive; for the symptoms of fever have subsided, the temperature has fallen to normal, there is no longer delirium or restlessness, the pulse rate is lower than before, and apparently the patient is beginning to recover.

Cervical cellulitis, if at all extensive, is very unfavourable; it leads to sloughing of the skin and subcutaneous tissue, and exposes the patient to the risks of exhaustion,

septicæmia, pyæmia, or hæmorrhage. If the cervical cellulitis accompanies severe faucial complications the case is nearly always hopeless, but when it occurs by itself a more favourable view may be taken.

Bronchitis and *lobular pneumonia* are serious complications when they arise in very young children.

The prognosis of *rheumatism* is good ; it rarely leads to chronic heart disease.

Of complications that come on during the period of convalescence the most grave is *diphtheria* ; for the fatality is high, and the larynx is frequently and early involved.

Otitis is dangerous because it may give rise to pyæmia, thrombosis of the lateral sinus, etc.

Nephritis occurs quite independently of the nature of the attack of scarlet fever. It is found most often in children from five to nine years of age, and somewhat more frequently in males than in females ; it is also more fatal in the former sex. The age of the patient is of importance from the point of view of prognosis, for the nephritis case-mortality is much higher for those under five years of age than for any other period of life.

One judges of the severity of this complication chiefly by the amount of urine that is excreted ; as long as it remains scanty the patient is in jeopardy. Coma is an unfavourable symptom, convulsions less so. If, when the acute febrile symptoms that so often mark the onset of the renal lesion have passed off, the urine still contains much albumen, recovery is likely to be tedious. Cases of nephritis supervening upon an unrecognised attack of scarlet fever are often severe and prolonged, because in most instances the nephritis has been neglected, not having been discovered till the onset of dropsy.

Scarlatinal nephritis usually ends in recovery ; but

sometimes we meet with a chronic renal disorder arising directly out of the acute attack.

The existence of *previous disease* in the person attacked by scarlet fever has often to be considered, both with respect to the course the fever is likely to run, and to its effect upon the already existing complaint.

All lung affections are unfavourably influenced by scarlet fever, and in the subjects of tuberculous disease recovery from scarlet fever is tardy, complications are common, and the tuberculous lesion, whatever it may be, often becomes rapidly worse.

Though usually much anxiety is caused when a person who is suffering from chronic heart-disease is attacked by scarlet fever, yet on several occasions we have been surprised to see the subjects of aortic regurgitation, or of mitral stenosis, pass through severe attacks of scarlet fever with no alteration in their cardiac symptoms.

Treatment.—In all cases the local affection of the fauces calls for active treatment of an antiseptic nature, in order to prevent the secondary invasion of the throat by the bacteria already mentioned. In mild cases a gargle or spray of permanganate of potash (fifteen grains to an ounce of water) or chlorine will be found sufficient. The latter is made as follows. Put four drachms of chlorate of potash into a dry bottle, and then pour in ninety minims of pure hydrochloric acid; chlorine gas is evolved, the evolution being hastened by shaking the stoppered bottle. When no more gas is given off gradually add thirty ounces of water. The water should be added a few ounces at a time, the bottle being shaken after each addition, so that the chlorine gas is dissolved in the water and not displaced from the bottle. Just before use one ounce of syrup should be added to every five ounces of the solution.

In more severe cases the following solution used as a spray will be found effective:—

Liniment of iodine	℥ xl.
Pure liquid carbolic acid	℥ ij.
Rectified spirit	℥ ss.
Glycerine	℥ iv.
Water to	℥ viij.

Another good antiseptic is perchloride of mercury, of which a solution of 1 in 1,000 to 2,000 parts of water may be used as a spray, but with caution on account of its poisonous nature.

The frequency with which these sprays are to be used depends upon the condition of the fauces. In cases with much exudation and œdema it may be necessary to make use of one or other of them every hour; in mild cases every four hours, or three times a day, is sufficient. It is difficult to spray the fauces of some children on account of the strenuous resistance they offer; in such cases the following powder may be blown upon the parts with benefit, though it is not so efficient as spraying or irrigation:—

Bicarbonate of soda	2 parts.
Chlorate of potash	1 part.
Borax	1 part.
Powdered white sugar	1 part.

In cases where there is much sloughing, injections of a few drops of a 1 in 20 solution of carbolic acid into the mucous membrane have been found to do good. These are given by means of a syringe fitted with a long needle, such as is used for exploring, but having a shoulder near its point to prevent too deep penetration.

The fauces may also be swabbed with pledgets of

cotton wool saturated with the solutions already mentioned, or with 1 in 40 carbolic lotion.

Lastly—and this is by far the most efficacious method of treating septic conditions of the throat—the mouth and fauces may be flushed or irrigated with a saturated solution of boracic acid, or the above-mentioned solutions of chlorine or permanganate of potash by means of a four-ounce ball, or a Higginson's syringe. In the case of a child the arms must be restrained by a sheet pinned around them, and the head fixed under the nurse's left arm, so that the mouth is over a basin, while the nurse works the syringe with the right hand. In cases where there is much prostration the child should be held on its side with its head on the edge of the pillow or mattress. The aid of a second nurse is often required for strong and vigorous children.

Pain may be alleviated by the use of cocaine (a 5 per cent. solution), either as a spray or applied to the fauces by means of a camel's hair brush. This solution must not be applied too frequently, otherwise symptoms of cocaine poisoning may arise. Hot fomentations or poultices to the neck often relieve pain; in some cases the sucking of ice has the same effect.

A high temperature without other symptoms rarely requires treatment, but a high temperature maintained for many hours, and accompanied by restlessness or delirium, calls for interference. By far the best method of treatment is the wet pack. The sheet should be wrung out of tepid water, and the patient kept in the pack for twenty or thirty minutes (see p. 12). Even when the temperature is but little reduced by this procedure (and in scarlet fever the temperature is very difficult to reduce) the patient will often fall into a refreshing slumber, and delirium will

cease. Sponging with tepid water will occasionally have the same effect.

Antipyretic drugs are less efficacious. The best is antifebrin (acetanilide), in doses of from two to ten grains, according to the age of the patient. It dissolves readily in rectified spirit. Water should not be added to the solution until just before it is to be administered, otherwise precipitation may take place. Here again the beneficial effect is often rather in the form of sleep and quietness than in any considerable reduction of temperature. It is necessary in some cases to repeat the dose two or three times, at intervals of three or four hours.

Delirium may require treatment. The wet pack, or a dose of bromide of potash and chloral hydrate or sulphonal, are usually sufficient. Opium is to be given with caution.

There are no known drugs that will either cut short an attack of scarlet fever, or, as has been alleged by some writers, prevent susceptible persons from being attacked. Belladonna and digitalis have little—in many cases no—beneficial effect in a bad case during the acute stage of the illness. A mixture containing strychnine or nuxvomica, bark and ammonia, may be given with advantage as a stimulant in cases where there is much prostration, or when prostration is apprehended; another excellent stimulant is spirits of camphor, in doses of from ten to fifteen minims every two, three, or four hours. Brandy (in moderate doses) and good champagne may also be given; the latter is especially useful when there is vomiting. In very severe cases, where there is difficulty in swallowing, ten to twenty minims of æther may be injected subcutaneously.

Attention to the diet and the surroundings of the patient are of importance. During the acute stage, milk—which

in the case of children should be diluted with water—or bread and milk are sufficient. It is advisable not to let the patient drink too greedily of the milk ; a few ounces should be given at intervals. Vomiting is best met by peptonising the milk. Five ounces of milk are added to a pint of water, and the mixture is heated to 140° Fahr. Two fluid drachms of liquor pancreaticus and twenty grains of bicarbonate of soda are then added, and the mixture kept at a temperature of about 130° Fahr. for half an hour, at the end of which time it is boiled for a minute or so. When sufficiently cool it is ready for use. Peptonised milk is not, however, pleasant to the taste. In severe cases the patient is too ill to appreciate flavours ; but when he can do so, the various peptonised foods that are now in the market may with advantage be substituted for milk peptonised as above directed. Some patients, adults as well as children, but chiefly the latter, obstinately refuse to take nourishment, no doubt on account of the pain experienced in attempts to swallow. They must be fed every four or six hours, by means of a nasal tube. Gruel, arrowroot, and chicken or mutton broth may also be given in addition to the diet mentioned above. It is best to keep the patient on slop diet until the temperature has become normal. Then fish may be allowed, with an egg and pudding (rice, custard, sago, etc.). A few ounces of fresh green vegetables are with advantage also added. After a few days chicken and then meat may be taken.

The length of time during which the patient is to be kept in bed will vary in different cases. If the temperature has been normal for a week, and the patient feels strong enough and is not the subject of any serious complication, there is no reason why he should not sit up for a few hours every day at the end of that time. The number of hours

during which he is allowed up daily may gradually be increased, and in warm and fine weather he may be allowed to go out of doors for a short time at the end of another week, provided that he can do so without risk of infection to other persons.

As soon as the patient is able to get out of bed he may be allowed to have a warm bath every evening. This will hasten desquamation, and relieve irritation of the skin. There is no necessity to oil the patient unless he be peeling freely, and it is desirable to keep the flakes and scales from being conveyed beyond the room in which he is confined.

A complication that calls for most careful treatment is *nephritis*. Inasmuch as this may set in very insidiously, it is advisable to test the urine for albumen frequently, at least every other day. It is during the third and fourth week that this complication most commonly arises. On the very first symptoms of nephritis the patient should be sent back to bed and put upon a farinaceous diet. In mild cases this treatment may be enough, but if there is scantiness of urine with symptoms of uræmia, it is advisable to employ diuretics. Of these the most serviceable are digitalis and citrate of caffeine, but the former drug is contra-indicated when the arterial tension is raised. The patient may also be allowed to drink as much barley water or "imperial drink" as he likes. The latter is made as follows: Two drachms of acid tartrate of potash are placed in a large jug, the juice of one lemon and some sugar or syrup are added, and two pints of boiling water poured in, the ingredients being well mixed. When cool, the drink may be placed by the patient's bedside, so that he can drink when he pleases. Linseed meal poultices should be applied to the loins

every three or four hours. If, under such treatment, there is no improvement in a few days, a warm bath (water at a temperature of 105° to 110° Fahr.) may be given every evening, the patient being kept in the bath thirty or forty minutes, and wrapped up in warm blankets on returning to bed. If the patient falls into a comatose state, dry cupping, or the application of leeches to the loins, should be tried. Convulsions may be similarly treated, and if severe may be checked by the inhalation of chloroform. Venesection to four or five ounces will sometimes cut them short. Pilocarpin must be used with caution in the acute scarlatinal nephritis of children. The same remark applies to the vapour bath.

Improvement is shown by an increase in the amount of urine passed, a decrease in the amount of albumen, and the disappearance of blood. When such a stage of improvement is reached fish may be added to the diet. If the improvement is maintained for a week or ten days a little meat and vegetables may be allowed, and gradually other articles of food. Some preparation of iron is with advantage prescribed ; *syrupus ferri phosphatis compositus* or the citrate of iron and quinine. The patient should not be allowed up as long as there is much albuminuria ; but in cases where the albuminuria has existed for several weeks it will often be found advantageous to let him get up and walk about. In some such cases it would seem that keeping the patient in bed maintained the albuminuria. Careful attention should be paid all through to the state of the bowels, so as to secure their regular action. The best purges are Epsom salts and sulphate of soda ; should these fail to act, compound jalap powder, castor oil, or warm water enemata will often suffice. Frequent vomiting in nephritis is best treated with bismuth, in

severe cases in conjunction with the use of nutrient enemata, so that the stomach is kept at rest.

Adenitis is treated by the application of linseed meal poultices or glycerine and belladonna. If suppuration takes place the abscess must be opened and treated on ordinary surgical principles. It is not necessary to make a large opening, nor to insert a drainage tube in most cases; it is sufficient to make a puncture with a scalpel, to express the pus, and apply boracic fomentations. As the majority of these abscesses occur in the neck it is desirable to avoid much scarring.

Cervical cellulitis is best treated with hot boracic fomentations. Making incisions early will very rarely prevent sloughing of the skin and subcutaneous cellular tissue. It is consequently advisable to wait till the skin has broken before incising, when it will usually be found to be undermined, and should be freely opened up. The underlying slough should be removed carefully; portions that have not separated from healthy tissues had better be left to come away by natural processes, as attempts at artificial removal often set up troublesome bleeding. The wound should be frequently irrigated with boracic lotion, and otherwise treated on the usual surgical principles.

Discharges from the nose and ears require the frequent syringing out of these cavities with a saturated solution of boracic acid in water. In the case of the ears, if this solution does not stop the discharge, or if the discharge becomes offensive, the following lotion will be found useful:—

Sulphate of zinc	2 grains
Carbolic acid	10 grains
Water	1 ounce,

or a solution of nitrate of silver, five grains to the ounce,

will often be successful. After every syringing the ear should be dried as thoroughly as possible with salalembroth wool, a piece of which should be placed in the external meatus.

A *mastoid abscess* is treated by an incision down to the bone; at the same time it is often advisable to freely open and clear out the mastoid antrum and cells. In a few cases it may be found necessary to explore the lateral sinus for thrombosis, or the brain for abscess. But for further information on these measures the reader is referred to surgical works.

The *earache* that precedes otorrhœa will be relieved by dropping into the ear a few drops of warm laudanum or cocaine solution, and covering the ear with hot cotton-wool, or by the application of leeches to the mastoid process.

For *rheumatism*, fifteen or twenty grains of salicylate of soda should be given every three or four hours, and the painful joints wrapped in cotton-wool. Under such treatment the symptoms quickly subside.

It is unnecessary to enter into the treatment of the remaining complications of scarlet fever, which is to be carried out on the usual medical and surgical principles.

It is possible that the antistreptococcal serum may be found to be beneficial in some of the septic complications.

CHAPTER VII.

DIPHTHERIA.

DIPHTHERIA is a disease due to the invasion usually of one of the mucous membranes by a specific micro-organism, the bacillus diphtheriæ. The mucous membranes most commonly affected are those of the throat and air-passages. The bacillus gives rise to a local inflammation, generally attended by the formation of a membranous exudation; and at the same time it produces certain toxic substances, the absorption of which causes many of the symptoms and complications of the disease. Of the latter a form of paralysis is the most striking.

Etiology.—The *geographical distribution* of diphtheria is at the present time very wide, and embraces Europe, North America, parts of South America, India, China, Australia, and South Africa. The disease is more prevalent in cold and temperate than in tropical climates.

It is only since the middle of the present century that we have had trustworthy data with respect to the occurrence and distribution of diphtheria in our own country. During the years 1858 and 1859 the registered mortality from this malady in England and Wales was

very high. Between 1861 and 1880 the mortality gradually fell to about a quarter of what it had been in 1859, but since 1880 a considerable increase has taken place. Up to the year 1880 the disease chiefly existed in rural districts; but since that date it has spread to the large towns, and notably to the metropolis. At the present time it is more prevalent in urban than in rural districts.

Season.—There is evidence to show that in England the mortality from diphtheria is highest during October, November, and December, and that the disease is more prevalent in those months.

Dissemination.—There is little doubt that infection from individual to individual is a most important factor in the spread of diphtheria. The virus may be conveyed from one person to another by direct contact, as in kissing, or indirectly by means of spoons, drinking vessels, etc. Those in attendance upon the sick have frequently become infected by the virus having been coughed or spat into their faces. It is also possible that the poison is conveyed in some instances by the breath.

The aggregation of large numbers of children in schools and similar institutions is one of the means by which the disease becomes disseminated.

In several epidemics milk has been proved to convey diphtheria. As in scarlet fever, in some instances the milk has become infected from a human source, but in others no such origin of infection has been traced. An eruptive disease of the udders of the cows from which the milk was obtained has in some of these latter cases been found to be present; and there is evidence to show that inoculation of healthy cows with diphtheria bacilli

will produce a similar eruptive disease. But this question is still under investigation.

It is possible that some domestic animals, especially cats and birds, suffer from diphtheria, and communicate it to human beings. Although the evidence in favour of such an infection is suggestive, it is by no means conclusive.

Defects of drainage have been considered by some writers to be potent factors in the causation of diphtheria. But most recent observers are of the opinion that the influence of such defects has been much exaggerated. Whereas during the past twenty years or so there has been a vast improvement in the sanitation of the country generally, yet diphtheria has been during that period, and apparently still is, on the increase; while enteric fever, the disease *par excellence* of evil drainage, has diminished.

There is no evidence of the dissemination of diphtheria by the water supply.

Continual dampness of soil appears to favour its development.

Diphtheria often occurs as a complication of other infectious disorders, especially measles and scarlet fever.

The number of cases of diphtheria notified in London in the year 1893 was 13,712, with 3,197 deaths. The case-rate was 3·2 per 1,000, and the case-mortality 23·3 per cent. In 1891 and 1892 the case-rate was 1·5 and 2·0 per 1000, and the case-mortality 22·5 and 22·2 per cent. For 1894 the case-rate was about 2·5 per 1000, and the case-mortality about 23·5 per cent.

Age.—Diphtheria especially attacks children under the age of ten. The following table shows the numbers and ages of the patients admitted into the Hospitals of the

Metropolitan Asylums Board during the years 1888 to 1894, with the number of deaths and fatality per cent.:—

Ages.				Number admitted.	Deaths.	Fatality per cent.
Under 1		199	123	61·8
1 to 2		688	434	63·1
2 „ 3		966	532	55·1
3 „ 4		1,259	608	48·3
4 „ 5		1,323	516	39·0
Total under 5				4,435	2,213	49·9
5 to 10		3,723	1,046	28·1
10 „ 15		1,330	141	10·6
15 „ 20		782	34	4·3
20 „ 25		543	25	4·6
25 „ 30		354	19	5·4
30 „ 35		183	9	4·9
35 „ 40		110	5	4·5
40 and upwards		138	24	17·4
Totals				11,598	3,516	30·3

From this table it will be seen that 8,158 out of 11,598 patients were under ten years of age. The table also exhibits the fatality. This was highest in infants under two years, and of the patients under five close on half died. The fatality falls after the tenth year, and remains low up to the fortieth, after which it again rises.

Diphtheria therefore resembles scarlet fever especially in attacking children under ten years of age, and in furnishing a much higher percentage fatality amongst those under five than in older patients. It differs from scarlet fever in two points. Firstly, it is more malignant, particularly amongst children. Secondly, the cases occurring in the first quinquennial period of life are about equal in number to those in the second quinquennial period; while in scarlet fever, as will be seen by referring to the

table on p. 59, the cases between the ages of five and ten are many more than those under five.

Sex.—Of the 11,598 patients in the table, 5,245 were males, and 6,353 females. The fatality of the former was 32, of the latter 29 per cent.

The **Incubation period** is usually from two to four days. It may perhaps be in some cases longer, but is always under seven days. It is occasionally shorter, especially in cases where direct inoculation has occurred. Thus in the case of Valleix, infected by a patient who coughed into his mouth, there was exudation on the tonsil on the following day (Trousseau).

Clinical History.—There are many gradations of severity, from the very mildest cases, in which there is little or no local exudation, while constitutional symptoms are slight or absent, up to the most grave, in which there is much thick membrane covering the fauces and severe toxic symptoms. A classification may also be made of the forms of diphtheria according to the part primarily affected, fauces, larynx, nose, etc. As, however, by far the most common seat of the local affection is the fauces, we shall describe the different forms of the disease as it appears in these parts, adding special observations on those cases in which the nasal, laryngeal, and other mucous membranes are the seat of the exudation.

FAUCIAL DIPHTHERIA.

The first symptoms of the faucial variety are usually sore throat, difficulty or pain in swallowing, malaise, and fever. Occasionally there may be vomiting, rarely a rigor. In some cases, especially in children, no complaint

is made of sore throat, and it is only on inspection that anything wrong is found.

The subsequent course taken by the disease depends upon the form it assumes, whether mild or severe. The degree of severity is partly dependent upon the degree of the local lesion, and partly upon other conditions, such as the constitution and age of the patient. It will be convenient to describe three forms of faucial diphtheria, the basis of the division being the degree of severity of the constitutional symptoms. These forms are the *mild*, the *moderately severe*, and the *severe* or *malignant*. We again remind the reader that there are all gradations between the mildest and most severe cases; and also that though at the commencement of an attack the disease may seem to be of a mild type, it may later assume a grave or malignant form. The local lesions may conveniently be described first.

Condition of fauces.—Every grade of severity may exist, from a simple angina without exudation, up to the formation of thick and extensive membrane. In the mildest forms the fauces are slightly swollen and reddened, and there is usually a little thin exudation present; but there may be no exudation at all. The exudation varies in its position and in its character. As to position, it may be situated on one or both tonsils, on the soft palate or uvula, or on the back of the pharynx. The tonsils are the parts most frequently affected. It is uncommon, while the tonsils remain free, to find the uvula or soft palate the seat of exudation. In severe cases the whole of the fauces are covered with membrane, which often spreads backwards over the walls of the pharynx. The character of the exudation varies greatly. It may be quite thin, and form a translucent layer; or

it may be thick, and then either of a pultaceous or gelatinous consistency, or of a leathery membranous nature. The exudation is often patchy in its distribution, but frequently forms a more or less uniform layer. The colour varies from dull white or yellow, to brownish-red, or almost black, from an admixture with blood. The membrane, especially when it is thick, is found to be very adherent, and, when forcibly removed, leaves behind a raw, bleeding surface.

The underlying and surrounding mucous membrane is moderately swollen, but there is not, as a rule, the same amount of swelling as is seen in scarlatina anginosa, although in a few cases there is marked œdema.

Sometimes superficial ulcers, covered with a thin pellicle, are seen, while now and then the exudation takes the form of yellow spots scattered over the tonsils, giving the appearance of a follicular tonsillitis.

Sloughing of the mucous membrane is uncommon, but this does at times occur, and leads to much loss of tissue. There is usually in the moderately severe, and even in some of the mild cases, a peculiar and offensive odour about the fauces, and in severe cases the odour is of a horribly fœtid character.

Pain is not a prominent feature, yet at times it may be agonising when any attempt is made to swallow.

When the disease terminates in recovery the membrane softens, becomes diffuent, and gradually disappears, leaving the mucous membrane finely granular; but in a few days the latter resumes its normal appearance.

The submaxillary and cervical *lymphatic glands* are usually enlarged, tender, and hard. When the affection of the fauces is severe the glandular enlargement is more marked, and there may be inflammation of the surrounding

connective tissue, infiltrating the skin and matting it and the underlying structures together.

The *pulse* is increased in frequency and is compressible even in many of the mildest examples of the disease.



CHART F.

Female, aged 8. Diphtheria, severe from the third day. There was not much exudation till the third day; after which there was a gradual increase, with toxic symptoms, till death.

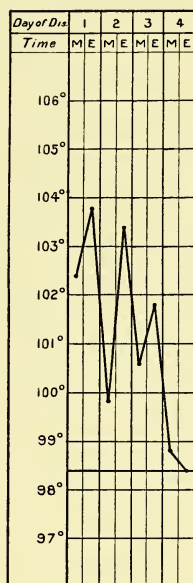


CHART G.

Female, aged 30. Mild, uncomplicated case of diphtheria, with irregular pyrexia. Exudation not gone till seventh day, but temperature normal after fourth.

In severe cases it becomes very frequent, and may be irregular.

The *temperature* is, as a rule, raised at the beginning of the attack. The height to which it rises varies, but it may reach 104° or 105°, although this elevation is not maintained for

any length of time. The course of the temperature is irregular, and gives very little indication of the severity or progress of an uncomplicated case. Sometimes, even in severe cases, there is no rise of temperature.

Albuminuria is so common in diphtheria that it may be regarded rather as a symptom than a complication of the disease. The frequency with which it occurs varies. It may be found in as many as three-quarters of the cases; it rarely occurs in less than one-quarter. It is most commonly to be detected about the fourth day of the disease, but it may occur as early as the first. Its appearance is not often postponed beyond the end of the first week. The amount and duration vary much, from a faint trace lasting for a day or two to a large quantity persisting for weeks. In the same case, too, the amount will often vary greatly from day to day. As a rule it may be said that the more severe the case the earlier does albumen appear in the urine, the greater is the quantity, and the more persistent its duration. The urine is usually of a normal colour, and there is little or no deposit. If there be pyrexia, urates may be deposited in abundance. Microscopically a few renal epithelial cells, and now and then a cast, may be found, but no blood cells; and there is no clinical evidence of an acute nephritis. In the large majority of cases the albuminuria completely clears up within a few days or weeks.

Anuria.—There is a certain class of case in which the action of the kidneys is seriously impaired. In such cases (usually, but not always, severe from the beginning) the urine, which contains a considerable amount of albumen, diminishes in quantity, so that only a few ounces are passed in the twenty-four hours. It remains quite clear, and does not contain blood, nor, as a rule, many casts.

These cases almost invariably end fatally. Death is often preceded by a period of total suppression of urine of one to three days' duration. In addition to the diminished excretion of urine the main symptoms are pallor, repeated vomiting, and a frequent and often irregular pulse. In a few cases there is, however, a marked infrequency of the heart's action. The patient is generally clear in his mind up to the last, though he may be very drowsy. At the *post-mortem* examination the kidneys appear normal to the naked eye; but microscopically fatty degeneration is sometimes found, and in a few instances there is a slight infiltration of the interstitial tissue with leucocytes.

An *acute nephritis*, with blood and casts in the urine, does occur sometimes, though rarely. It was observed five times in 1,071 consecutive cases at the Eastern Fever Hospital.

TYPES OF FAUCIAL DIPHTHERIA.—The course of an attack of diphtheria and the constitutional symptoms depend upon the type assumed.

In the *mildest cases*, in which there is little or even no exudation upon the fauces, constitutional symptoms are very slight or absent. In the *mild form*, where the exudation, though present, is of limited extent, affecting only the tonsils, the symptoms consist of moderate and transient pyrexia and slight malaise, with little, if any, enlargement of the cervical glands. The exudation clears off in two or three days; albuminuria, if present, lasts but a short time, and the patient is well in a week or ten days. But it is to be remembered that even in the mildest form there is risk of the larynx becoming affected, of cardiac failure ensuing, and of paralysis, sometimes fatal, arising at a later period.

In the *moderately severe form* the throat symptoms are more pronounced, and the exudation is seen upon the uvula and soft palate as well as upon the tonsils. Pyrexia is often marked, the temperature rising to 104° or 105° Fahr. on the first day, but it may be slight. The pulse is frequent and compressible, the tongue furred, and the face pallid. The cervical glands are enlarged, but not fixed by surrounding inflammation. There is often a discharge from the nose, and albuminuria sooner or later makes its appearance. It is usually at least a week before the faucial exudation clears off, and the patient begins to convalesce. Should the case progress favourably, restoration to health will take another fortnight or three weeks. There is always, however, danger of the onset of cardiac failure, even for some time after the exudation has disappeared. The pulse becomes very frequent and irregular, and death may occur quite suddenly; occasional marked infrequency of pulse is observed. Again, suppression of urine may set in, or the larynx become involved. In some patients the faucial exudation increases, and the case passes into the severe form, and ends fatally. Subsequent paralysis is comparatively frequent in this form of the disease.

In the *very severe* and *malignant* forms there is much exudation, and the constitutional symptoms are grave. A sheet of membrane is to be seen covering the veil of the palate, and stretching across from one pillar to the other, completely hiding the pharynx beyond. Prolongations of the membrane extend backwards to the pharynx. An ichorous discharge, often blood-stained, runs from the nose, and a peculiar and very fœtid odour is given off. There is considerable inflammation of the cervical glands, and even of the surrounding cellular tissue. The tempera-

ture is usually raised, especially when any inflammatory complications are present; still it is not uncommonly normal or subnormal. Prostration is an almost invariable feature of the grave forms of diphtheria. The heart's action is frequent—120 to 140 or more per minute—the pulse is compressible, the skin is pale, and often of a yellowish waxy appearance. There is restlessness, but rarely that active delirium which is observed in severe cases of scarlet fever. Drowsiness and apathy are common. The patient resents being disturbed, and refuses food. The mind remains unclouded up to the very end. There is much albumen in the urine. Death usually occurs within a week from the onset. Should the patient survive this period he is extremely likely to fall a victim to one or other of the complications which are described below. The fatal termination takes place, sometimes very suddenly, by cardiac failure, and may be preceded by convulsions.

In a few of the most severe cases there is hæmorrhage from one or more of the mucous membranes, from the nose, gums, pharynx, stomach, intestines, vagina, and kidneys. A purpuric eruption may appear on the trunk and limbs, and extravasations of blood take place in the subcutaneous and other cellular tissues. Such cases may be described as *hæmorrhagic diphtheria*.

OTHER FORMS OF DIPHTHERIA.

Other mucous membranes besides that of the fauces may be the seat of the diphtherial process. The nasal passages, larynx, trachea, and bronchi are very commonly implicated. So also, but much more rarely, are the mouth, lips, stomach, vulva, prepuce, conjunctivæ, and the external auditory canal. Usually when exudation is to be

seen in these places it is also present upon the fauces. But the diphtherial process may commence in and be limited to the larynx and trachea, nasal cavities, mouth, conjunctivæ, or genital organs, the latter being more frequently affected in females than in males. The inner surfaces of the labia majora become covered with a dirty-grey membrane, and have a sloughy appearance. The genitals become swollen, tense, red, and painful. The disease is usually limited to the external genitals, although extension along the vagina to the uterus may follow. The skin also may be affected by diphtheria, but this condition is rare.

NASAL DIPHTHERIA.—Attention is usually directed to the presence of this variety by a discharge from the nose, thin and watery, or thick and muco-purulent. It is often mixed with blood, and epistaxis is of frequent occurrence. In prolonged cases the anterior nares become excoriated. Shreds of membrane or casts of the nasal fossæ may be obtained by syringing, or may be spontaneously discharged. It is uncommon to see membrane lining the anterior nares. There is no doubt that diphtheria may begin in and be limited to the nasal cavities. These cases are often unrecognised, the constitutional symptoms are generally mild, but the disease sometimes runs a very prolonged course.

LARYNGEAL AND TRACHEAL DIPHTHERIA.—*Croup*.—Faucial is very often associated with laryngeal diphtheria. When this occurs the clinical aspect of the case alters, for the predominant symptoms are those due to the obstruction of the respiratory tract, *i.e.* to "*croup*." Now much confusion has arisen in connection with this term, and it will be convenient to say a few words in this place concerning its use and signification. The term is

used by us entirely in a clinical sense. From this point of view there is no such disease as "croup." By "croup" we mean obstruction of the air passage at or about the larynx, giving rise to dyspnœa; and this may be due to more than one cause, the presence of membrane, a foreign body, and so on. So that in any case of "croup" that comes before him the practitioner should ask himself, "What is the cause of the obstruction?"

Laryngeal diphtheria is usually secondary to diphtheria of the fauces, but it may occur quite independently of the latter. Formerly there was much discussion as to whether a membranous laryngitis, occurring without the fauces being affected, was true diphtheria or not. There is now sufficient bacteriological evidence to show that diphtheria may primarily affect the larynx and leave the fauces free. On the other hand, there is no doubt that a membranous laryngitis may occur quite apart from diphtheria. A few cases of this nature have been carefully examined in which the diphtheria bacillus has been proved to be absent. Nevertheless these cases are rare, and it is best to look upon all cases of membranous laryngitis as diphtheria, unless there is distinct bacteriological evidence to the contrary.

Although laryngeal diphtheria is usually secondary to diphtheria of the fauces, it is by no means uncommon for laryngeal symptoms to be the first from which the patient suffers. "Croup," then, is frequently the first and most prominent symptom of diphtheria, and it is only by examination that the fauces are found to be affected.

The invasion of the larynx by the diphtherial process usually occurs within the first week of the disease. But in the prolonged form of diphtheria this event may be postponed, even to beyond the fourth week. As long

as exudation remains upon the fauces there is a risk of it spreading to the air-passages. The earliest symptom of this complication is a frequent cough of a harsh metallic nature. Soon succeed loss of voice (so that the patient can speak only in a whisper) and stridor. All these symptoms may come on very rapidly. After they have become established one of three things happens.

Firstly, paroxysmal attacks of inspiratory dyspnœa may occur. In these attacks there is laboured inspiration, recession of the lower portion of the thorax, of the epigastric angle, and of the space just above the sternum. The patient becomes cyanosed, is exceedingly distressed, and, in his anguish, tears at his throat, clothes, and whatever may be near. The face is anxious, the eyes protrude, and the skin is bathed in perspiration. In most cases the attacks are not at first very urgent, but they become so after three or four paroxysms; and should relief not be afforded the patient may die, either from dyspnœa or from exhaustion. Between the attacks there is much relief, and the patient may even fall asleep, although there is not absolute cessation of the symptoms. This occurs especially when the paroxysm has ended in a fit of coughing resulting in the expulsion of membrane.

Secondly, symptoms of inspiratory dyspnœa may come on gradually. Inspiratory stridor, cyanosis, and recession are slowly established, usually without the occurrence of any urgent attacks, and prostration ensues. The patients do not seem to suffer to the same degree as in the cases described in the preceding paragraph. This form of the disease is met with especially in adults, in whom membrane may line the larynx, trachea, and even the larger bronchial tubes, with but little dyspnœa becoming apparent. But when the membrane begins to form in

the smaller tubes, and the normal exchange of gases is impeded, dyspnœa sets in, and the patient quickly dies.

Thirdly, the symptoms of laryngeal implication may never become urgent, even though membranous casts of the larynx, trachea, and large bronchi be coughed up, and the patient recovers without operative interference, or in toxic cases dies of the severity of the disease by cardiac failure.

In four cases that have come under our observation patients have expectorated membranous casts of the trachea and bronchi without suffering from dyspnœa, except very slightly at the time of expulsion of the casts. Perhaps in these cases the larynx escaped the diphtherial process. In all the fauces were affected. One patient died from prostration without dyspnœa. The remaining three subsequently became paralysed, but ultimately recovered.

WOUND DIPHTHERIA.—In rare cases the diphtherial process primarily affects the subcutaneous tissue, the infection generally occurring through some trivial puncture or abrasion of the skin. A superficial abscess or a small ulcer discharging a thin secretion is the result. The ulcer frequently heals almost completely, and then breaks down again, and may in this way continue for a long period. Sometimes a distinct membrane forms, and there may be much œdema of the surrounding parts. The authors have had under their notice cases of primary diphtheria of the fingers, in which the diphtheria-bacillus has been found. In some of the cases the fauces have ultimately become affected, no doubt through the direct conveyance of the virus to the mouth of the patient. This, indeed, constitutes the principal danger, for the constitutional symptoms of this form of diphtheria are usually, though not invariably, mild.

THE PROLONGED FORM OF DIPHTHERIA.

We have already stated that in moderately severe cases of diphtheria the exudation is present upon the fauces for at least a week. And cases are by no means uncommon where membranous shreds and casts are repeatedly detached from the fauces, nasal passages, and even the larynx and trachea, for two, three, and occasionally four weeks. But in the prolonged form of diphtheria membrane is continually being re-formed for a period of time much longer than this. Cadet de Gassicourt relates cases, from the experience of himself and others, in which the membranous formation persisted for periods of time ranging from six weeks to nine months.

At the commencement such cases differ in no respects from the ordinary forms of diphtheria. But instead of resolution taking place within three or four weeks, the disease settles, as it were, on one of the mucous membranes, more especially that of the nasal passages, and persists for a long period. In some cases death is the result, and this termination is observed more particularly in those laryngeal cases where tracheotomy has been found necessary at an advanced period of the disease.

Complications.—By far the most important complication is *paralysis*. Sometimes it comes on so long after the attack of diphtheria as to be regarded rather as a sequel than a complication. It occurred in nearly 12 per cent. (125 out of 1,071) of the cases of diphtheria, including those fatal at an early period of the disease, admitted into the Eastern Hospital during the years 1892 and 1893. Excluding these fatal cases, the incidence of paralysis was nearly 18 per cent. With regard to age, it was found that, excluding cases dying during the acute stage of

the attack of diphtheria, 22 per cent. of the patients under ten suffered from paralysis, 15 per cent. of those between ten and twenty, and nearly 4 per cent. of those over twenty. It may thus be stated generally that the younger the patient the more likely is he to be attacked with paralysis.

Paralytic symptoms most commonly begin to show themselves during the second, third, or fourth week of the illness. They may, however, appear during the first week, and as late as the end of the seventh, occasionally even later.

In most cases the *soft palate* is the part first affected, and the symptoms are a "nasal voice," and an escape of liquid through the nose when the patient drinks. Accompanying these there is often difficulty in swallowing, from a participation of the pharynx in the paralysis. On examining the throat the soft palate is seen to be motionless, partially or completely, when attempts at phonation are made. Sensation also is impaired or lost, for on touching the soft palate no reflex movements are set up in those cases in which the movement on phonation is not completely in abeyance.

The paralysis may progress no further, and in many cases does not. Often, however, other muscles become affected after a few days. The next to fail are usually the ciliary muscles, then those of the lower extremities, then one or more of the ocular muscles, the muscles of the trunk, neck, upper extremities, and of respiration, including those of the larynx, so that there is a more or less generalised paralysis. It is excessively rare for the muscles of the tongue or face to suffer.

It is not uncommon for the *ciliary* muscles to be the first paralysed, and to be the only ones affected. The

symptom pointing to cycloplegia, or ciliary paralysis, is a difficulty in accommodating the lens for near objects, and the patient is unable to read when a book is held at the usual distance from the eyes.

Paralysis of the *pharyngeal muscles* and of the *epiglottis* shows itself by difficulty in swallowing, and a fit of coughing is set up whenever the patient drinks. Solid food also may get into the larynx if care be not exercised. In some cases the paralysis is complete, and swallowing becomes impossible. Perhaps in these cases the œsophagus also is implicated. Food may get into the larynx when the mucous membrane is anæsthetic, even if there is no motor paralysis.

When the *lower extremities* are involved there is, in the first instance, unsteadiness of gait (ataxia), paresis, and loss of the knee-jerk. This condition may pass into one of complete motor paraplegia. So it is with the *upper extremities*, which, however, are not so often, nor so severely, affected as the lower. Paralysis of the muscles of the *trunk* and *neck* is shown by inability to sit upright, to turn over in bed, and to lift the head from the pillow.

When the *respiratory* muscles suffer there is deficient expansion of the thorax, and dyspnœa. Sometimes the *diaphragm* alone is paralysed, in which case the abdomen is motionless, or recedes during inspiration, when it should move forward, and the lower portion of the thorax is unduly widened from side to side.

When the adductor muscles of the *larynx* are involved there is aphonia, and the cough is ineffectual. In some rare instances only the abductors suffer, in which case there is stridor and inspiratory dyspnœa, but the power of phonation and of coughing is retained, though the voice is usually altered in character.

Of the *external ocular muscles* the most commonly affected is one of the external recti, though both may suffer ; as may, though much less commonly, one or both of the internal recti. It is not common to find other ocular muscles paralysed, but in rare cases there may be complete loss of movement of one or both eyeballs. The levator palpebræ is very rarely involved. It is only in extreme cases of generalised paralysis that the *iris* is affected ; and then its reaction both to light and accommodation is sluggish. The optic discs are always normal.

It is very rare to find the muscles of the *bladder* or *rectum* involved.

In some cases, usually those in which the palsy is widespread, the following symptoms come on : pallor, frequent vomiting, pain in the epigastrium or abdomen, greatly increased frequency and weakness of pulse (140 to 170), and irregular respiration. These are the most dangerous symptoms that can arise in the course of paralysis, for they almost invariably prove fatal. It is usual to ascribe them to an implication of certain branches of the *vagus*. These symptoms are rarely seen in cases where the paralysis is limited, as, for instance, to the palate. Still more rarely do they arise in convalescent patients apart from any other symptoms of paralysis. They may be met with, however, during the acute stage of an attack of diphtheria ; possibly in such instances the pathological condition is different, and is due to fatty degeneration of the cardiac muscle.

It has been stated that the symptoms of paralysis nearly always show themselves first in the palate or ciliary muscles, and that they may progress no further. But occasionally some other group of muscles (*e.g.*, those of the lower extremities, of the trunk, or of respiration) is

the starting point of the paralysis, which may remain localised to these parts.

While diphtherial paralysis shows itself chiefly in an impairment of motion, it must be mentioned that it is not at all uncommon to find *sensory disturbances*. Adults nearly always complain of numbness or of "pins and needles" in the extremities, even before motion is much affected. The tongue also may feel numb, and the soft palate be insensitive. If the skin be carefully tested, impairment of sensibility to touch and pain can often be made out in patients who are old enough to give intelligent replies. But, inasmuch as the loss of sensation is rarely, if ever, complete, it is exceedingly difficult to ascertain the presence of sensory disturbances in children, excepting in the case of the soft palate, through which a needle can often be thrust without causing either pain or reflex movement. The special senses are never impaired.

The condition of the *reflexes* and *tendon phenomena* varies. It is very common to find the knee-jerk lost even before there is distinct paresis of the lower extremities, and the same is true of the wrist and elbow jerks. In some cases for several days before the knee-jerk is diminished it becomes decidedly brisk. In exceptional cases it is exaggerated all through the attack of paralysis, provided the lower extremities are unaffected. It should be added that it is not at all uncommon to find the knee-jerk lost during convalescence from diphtheria, even in cases where no symptoms of paralysis arise. The knee-jerk, when abolished, is absent for some weeks, or even months; occasionally it will return for a time to disappear again later. The same is true, though it is not common, of the paralysis itself, especially in the case of the palate. The degree of paralysis may vary

from time to time. As for the superficial reflexes, they are not usually lost unless the parts with which they are concerned are affected to a considerable degree.

There is usually wasting and flabbiness of the muscles of the affected regions. In cases of generalised paralysis the patient may become emaciated ; he is often in such cases dull, listless, and apathetic. In slight cases there is no alteration in the electrical reaction of the muscles ; but in the more severe the reaction to faradaism may be impaired or lost, while in the most severe there is the "reaction of degeneration."

The duration of an attack of paralysis varies from a few days to many weeks or even months. The average time is from six to eight weeks. Recovery is almost always complete, and any permanent palsy is exceedingly rare.

There are several dangers attending paralysis. Solid food may get into the larynx and lead to asphyxia, while liquids may reach the lungs and set up lobular pneumonia. Should the respiratory muscles be paralysed, the patient runs a risk of dying from inability to expand the chest, or from the accumulation of mucus in the tubes. Death may also be due to sudden cardiac failure, especially in cases where there is reason to believe the branches of the vagi are involved.

Paralysis is most often observed following faucial diphtheria, for this is by far the most common form of the disease. But it may follow other forms, and cases have been recorded in which it has complicated diphtheria of the vulva and diphtheria of a wound. With respect to the different varieties of faucial diphtheria, paralysis may follow a mild primary attack ; but it does not follow a mild attack so often as a severe one, even when allowance is made for the fact that so many severe

cases of diphtheria prove fatal before reaching the stage at which paralysis usually appears. The worse the attack of diphtheria, the more extensive and persistent the exudation, the larger the amount, and the more persistent the presence of albumen in the urine, the more likely is paralysis to supervene, and, having made its appearance, to become generalised.

The cause of the paralysis is no doubt in nearly all instances the affection of the peripheral nerves, which will be found described in the section on the morbid anatomy of the disease (p. 137).

Cardiac complications.—During both the acute and convalescent stage certain dangerous cardiac symptoms are occasionally observed. We have already stated that in the course of a paralysis such symptoms may arise. But they are also met with quite apart from paralysis. The pulse becomes either very slow and irregular, or extremely rapid. The first sound of the heart is short. Death results from cardiac failure, sometimes quite suddenly without any previous warning, at others after an increasing failure of the pulse, with pallor and faintness, has given an indication of the critical state of the patient. These cases are probably due to fatty degeneration of the cardiac muscle. Occasionally with these symptoms dilatation of the heart may be discovered.

Lobular pneumonia is the most frequent cause of death in diphtheria of the air passages. An increase in the rate of breathing, a high temperature, and the presence of râles and perhaps of patches of tubular breathing, are the chief signs. An *enlargement* of the *cervical lymphatic glands* is common, and this sometimes results in suppuration. *Cellulitis of the neck* may also occur. *Otitis* and *otorrhœa* are not uncommon. *Lobar pneumonia* is not often

met with, while *pleurisy*, *peri-* and *endocarditis* are rare. *Renal complications* have already been described (see p. 118). Secondary *erythematous rashes* are occasionally seen.

Relapses.—A marked relapse was observed in 16 out of 1,071 consecutive cases of diphtheria admitted into the Eastern Hospital. A slight recrudescence of the sore throat is more common still. By a relapse we mean a fresh attack supervening before the patient has recovered from the effects of the primary attack. It may be worse than the first, and even prove fatal.

Dr. Gresswell has drawn attention to cases in which persons, after an attack of diphtheria, have become subject to attacks of sore throat, at times of an apparently mild character. These subsequent attacks have, however, been recrudescences of the original attack of diphtheria; for the individuals in question have, while suffering from them, been the source of severe and even fatal attacks of true diphtheria in persons with whom they have been brought in contact.

Protection.—It is uncertain how far one attack of this disease protects against a second. Certainly relapses and second attacks are by no means rare. Some authors, indeed, consider that one attack predisposes to another. This we doubt; and we think that one attack confers some, though not complete protection. Difficulties in diagnosis have perhaps led to the difference of opinion that exists on this point.

Length of Infectivity.—Until quite recently there were no reliable means of ascertaining the length of time during which a patient remained infectious, but now careful bacteriological examination will decide this question. As long as diphtheria bacilli are to be found in the secretion of the previously affected mucous membrane

the patient is infectious. The bacilli can be detected some time after the membrane has completely disappeared. Dr. Park examined 742 cases with the following results. In 315 the bacilli were no longer present three days after the complete disappearance of the exudation. In 427 cases the bacilli persisted for a longer time—viz., in 201 for five to seven days; in 84 for twelve days; in 69 for fifteen days; in 57 for three weeks; in 11 for four weeks; and in 5 for five weeks, or longer. The use of antiseptic irrigations of the throat and nose led to a much more rapid disappearance of the bacilli, but even then they sometimes persisted for as long as twenty days. When a bacteriological examination cannot be made, the patient should be considered infectious for at least three weeks after the exudation has completely disappeared.

Instances have recently been recorded in which virulent diphtheria bacilli have been obtained from the fauces several months after an attack of diphtheria.

Morbid anatomy.—Besides the exudation there is but little to be seen with the naked eye at a *post-mortem* examination on a case of diphtheria. When definite membrane is present, it is found to differ with respect to its adherence to the underlying mucous surface according to the part affected. Upon the palate and the adjoining structures the membrane is, as a rule, very closely adherent, and sometimes is with much difficulty removed with scalpel and forceps. The superficial layers of the mucous membrane may be in a necrotic state, and into the deeper layers blood may be effused. Anything approaching the deep ulceration commonly met with in scarlet fever is only occasionally seen. In the nasal fossæ the membrane, even when thick and tough, is loosely adherent.

In the larynx, trachea, and bronchi the membrane is usually very loosely adherent, and is of a whiter hue than that upon the fauces. In the trachea it is often marked with transverse depressions corresponding to the rings of that structure. The membrane becomes thinner and less tough towards the peripheral divisions of the bronchi, and is lost in thick mucus after the second, third, or fourth branchings. There is frequently superficial ulceration or excoriation of the mucous membrane of the larynx and trachea. In some cases, especially after tracheotomy has been performed, the bronchial tubes are full of muco-pus. Often there is extensive collapse of the lungs, or lobular pneumonia.

The microscopical appearances of the membrane are essentially similar wherever it is found. The membrane consists of masses of fibrin, either granular or fibrillated, with a few leucocytes scattered through them. The underlying mucous membrane is covered with a dense layer of leucocytes, and it is infiltrated with fibrin, leucocytes, and often red blood corpuscles. The epithelium has usually completely disappeared. In the most intense cases the tissues of the mucous membrane become necrosed, the cells break up, and their nuclei no longer stain with the usual reagents. In the trachea the membrane is more laminated, and is separated from the underlying tissue by a clearer line of demarcation than exists in the case of exudation upon the tonsils and palate. The diphtheria bacilli (see below) are only found on the surface of the exudation, and are separated from the mucous membrane by the masses and layers of fibrin. In some cases streptococci are found penetrating deeply into the mucous membrane. The whole process is that of a fibrinous inflammation produced by the

diphtheria bacillus. It must be observed that fibrinous inflammation of a similar character may be set up by other micro-organisms.

In cases where the exudation has not been membranous nothing more is visible to the unaided eye than has been observed during life. Indeed, in a few cases more evidence of the disease is apparent before, than after, death, even when this has occurred in the acute stage ; for any exudation which was present may have been removed by treatment. The cervical lymphatic glands are usually slightly swollen and of a pink colour. The same is true of the bronchial glands when the lungs are inflamed. Sometimes we find the mesenteric glands, and more rarely the lymphoid tissue of the intestines (Peyer's patches and solitary follicles), swollen. In all uncomplicated cases the remaining organs do not present any morbid change that the eye can detect. The spleen is not enlarged, and the kidneys usually appear normal. A microscopical examination will sometimes reveal fatty changes in the parenchymatous tissue of the liver and kidneys. In cases where death has occurred from cardiac syncope the heart-muscle will be found to be similarly affected.

When death takes place after the exudation has cleared off, whether it be due to syncope, suppression of urine, or paralysis, there is in most cases no macroscopic appearance to point to the cause of death. The aspect of the body and its organs may, in fact, be that of health. Wasting occurs only in cases where constant vomiting has existed for some days before death, or when death has been the termination of a long and severe attack of paralysis. Even in such instances the wasting is rarely extreme. In cases where death has been preceded by vomiting and suppression of urine, the skin is unusually

pale and anæmic, and the bladder is empty and contracted.

In the hæmorrhagic form effusion of blood may be found in the subcutaneous, retro-peritoneal and pharyngeal connective tissue, and sometimes into the voluntary muscles. Submucous hæmorrhages have also been observed in the stomach, intestines, bladder, and uterus. Hæmorrhages may also be seen in the walls of the heart.

When death has taken place during paralysis the microscope reveals a degeneration of the affected nerves. This degeneration is generally limited to the periphery, but it may extend along the trunk of the nerve to the anterior nerve root. The degeneration is parenchymatous, and consists of a disappearance of the white substance with some multiplication of the nuclei of the nerve sheath. In severe cases there is rupture of the axis cylinder. It is only in the nerves of the palate that an interstitial inflammation occurs. In some cases the corresponding cells of the anterior cornua undergo a fatty degeneration. This is probably secondary to the nerve degeneration.

The **bacillus diphtheriæ** is a non-motile rod-shaped micro-organism, which presents great variations in its morphology. In length it varies from 1·5 to 6 μ . It is often wedge-shaped, with one end drawn out into a fine point consisting of the sheath, and two bacilli are frequently united together with the bases of the wedge in apposition. The protoplasm often stains irregularly, giving a beaded appearance to the bacillus, but in young bacilli it stains uniformly. The former appearance is best seen in the long forms, and is well brought out in specimens stained with methylene blue. The rods are either straight or curved. It is common to find pairs of

bacilli grouped together with their long axes parallel ; but several may be grouped crosswise, or interlaced together so as to form rosettes. In older cultivations one or both ends of the bacilli are often swollen, producing club-shaped bodies. Short wedge-shaped bodies, exhibiting transverse striations, are not infrequently met with. The morphology depends, to a certain extent, upon the method of cultivation. Thus in some kinds of broth the bacilli are very short, while on glycerine agar and blood serum they are long. The appearance and length of the bacilli vary too with the age of the cultivation. Spore formation does not occur.

The bacillus can be stained by the usual aniline dyes, and is not decolorised by Gram's method.

The bacillus grows best at the temperature of the body, but it will grow at the ordinary temperature of the air. Cultivations can readily be obtained upon blood serum, agar, broth, and gelatine. The latter medium is not liquefied. On the surface of blood serum, at the body temperature, white opaque colonies of the size of a pin's head appear in twelve to eighteen hours. These colonies are unlike those of the majority of the bacteria met with in the mouth, and consequently this medium is used in making a bacteriological examination for the purposes of diagnosis.

Varieties of the diphtheria bacillus. *The pseudo-diphtheria bacillus.* *Xerosis bacillus.*— There are at least two morphological varieties of the true diphtheria bacillus which can be distinguished from one another by careful microscopical examination of cultivations made under exactly similar conditions. Besides other points of distinction a difference in length can be observed between these varieties ; and it is probable that they correspond to the " long " and " medium " varieties of the French authors.



Fig. 1.—Long Diphtheria bacillus.
Serum cultivation.
12 hours at 37° C.



Fig. 2.—Long Diphtheria bacillus.
Serum cultivation.
7 days at 37° C.

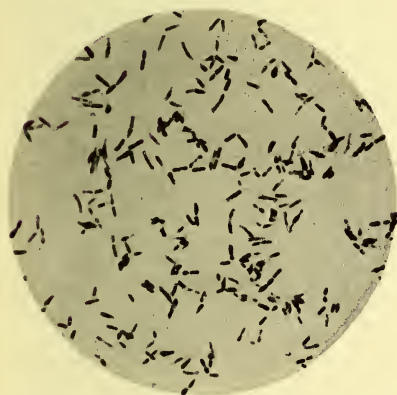


Fig. 3.—Short Diphtheria bacillus.
Serum cultivation.
24 hours at 37° C.



Fig. 4.—Short Diphtheria bacillus.
Serum cultivation.
14 days at 37° C.

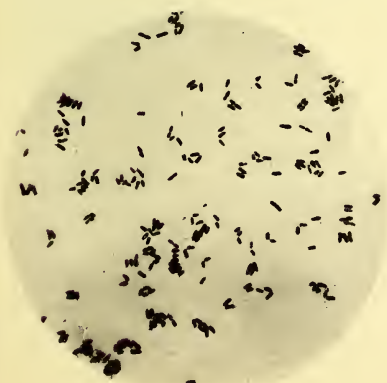


Fig. 5.—Hoffman's bacillus.
Serum cultivation.
12 hours at 37° C.

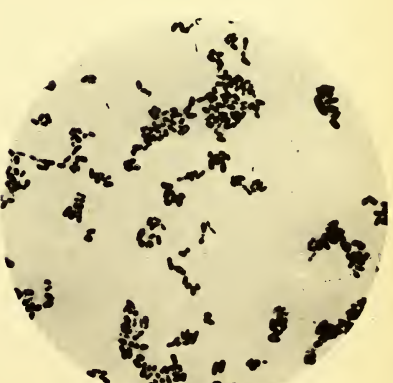


Fig. 6.—Hoffman's bacillus.
Serum cultivation.
7 days at 37° C.

In comparing these varieties care must be taken that the conditions of growth are similar; for, as has already been stated, the diphtheria bacillus presents different morphological appearances when grown on different media.

In addition to these varieties there is a bacillus which is sometimes associated with them in the throats of patients suffering from diphtheria, but which we believe bears no causal relation to the disease. This bacillus, the *pseudo-diphtheria bacillus*, was first described by Hoffman, and possibly corresponds to the "short" variety of the diphtheria bacillus of the French authors. It would be better to call it Hoffman's bacillus, because some observers designate every non-virulent form of the diphtheria bacillus by the name pseudo-diphtheria bacillus. Hoffman's bacilli are short, wedge-shaped rods, generally united in pairs with the bases in apposition. They stain uniformly, and do not show the same variations in morphology as the true diphtheria bacillus when cultivated on different media. In old cultivations clubbed forms are found which closely resemble those of the true diphtheria bacillus. The appearance of the colonies is almost absolutely identical with that of the diphtheria bacillus. The bacillus is not pathogenic to animals.

The *xerosis bacillus* is a bacillus which has been found in various inflammatory conditions of the conjunctiva, and which bears a close resemblance to the long variety of the diphtheria bacillus. It differs in being more curved and more frequently arranged in rosettes. In the first cultivation derived from the human body, made on blood serum at the body temperature, the colonies do not develop until the end of forty-eight hours. It is not pathogenic to animals.

The method of making a bacteriological examination in cases of suspected diphtheria is as follows. The tongue is depressed with a spatula, and a portion of the exudation is removed by scraping or rubbing over the fauces with a sterilised instrument. For this purpose either a platinum rod is employed, which can be sterilised in a flame just before use; or a plug of cotton-wool fixed on the end of a metal rod, previously sterilised, and kept in a sterilised test-tube plugged with cotton-wool. In laryngeal cases, if the fauces are not visibly affected, the instrument must be passed as far as possible downwards towards the larynx. Two or three blood-serum tubes are then inoculated by smearing their surface with the platinum rod or plug of cotton-wool. The tubes are placed in an incubator, and kept at the body temperature. At the end of twelve or eighteen hours the diphtheria colonies appear as opaque white round masses the size of a pin's head. On microscopical examination a diagnosis can be made. A portion of one of the colonies is removed with a sterilised platinum wire, is diluted with a drop of water, and spread over the surface of a cover glass. The cover glass is allowed to dry in the air, is passed three times through a flame, and is stained for five minutes in a solution of carbolic-methylene-blue, prepared in the following manner:—One and a half grammes of methylene blue are rubbed up in a mortar with 10 cc. of alcohol, and 100 cc. of a 5 per cent. solution of carbolic acid in water are gradually added. The cover glass is then washed with water, dried in the air, mounted on a slide with Canada balsam, and examined with a one-twelfth oil-immersion lens. Should the case be one of diphtheria, bacilli with the characters already described will be found.

If the exudation on the throat be examined directly under the microscope it will in all cases show a number of different kinds of bacteria. Many of these bacteria are the normal inhabitants of the mouth, and do not form colonies upon the surface of blood serum. Consequently in some cases of diphtheria the only colonies which develop in the tubes are those of the diphtheria bacillus. But in the majority of cases other colonies also develop. The most important of these are minute transparent colonies of a streptococcus similar to, if not identical with, the streptococcus pyogenes. We shall presently discuss the signification of the presence of this streptococcus.

We must add that a single bacteriological examination will not always detect the presence of diphtheria bacilli, even in cases of undoubted diphtheria. This may be due to the cultivation having been taken from an area free from bacilli, to the local use of antiseptics, or to some other error of examination. It is therefore sometimes necessary to make further examinations.

The value of a bacteriological examination.—We have already insisted upon the importance of making a bacteriological examination in all doubtful cases of diphtheria. It is of special value in detecting mild cases which are so often the cause of the spread of the disease. If diphtheria should break out in a school or in the wards of a hospital, a systematic bacteriological examination should always be made. Diphtheria bacilli will, under these circumstances, often be found in the throats of individuals who appear in perfect health, but who are nevertheless in an infectious condition; and the isolation of these persons will minimise the danger of a spread of the disease.

Pathology.—The cause of diphtheria is undoubtedly the diphtheria bacillus. Its presence can be demonstrated in the exudation from all cases of diphtheria.

Pure cultivations are pathogenic in the case of many animals, and when inoculations are made in a suitable manner the same symptoms are produced as in human beings. Guinea-pigs are especially susceptible. A small quantity of a cultivation injected under the skin of these animals produces a local œdema and death in a few days. Rabbits are less susceptible, and a larger dose is required to produce death. When death is delayed symptoms of paralysis often precede the fatal termination.

Typical membranous exudations, such as are found in the human subject, can be produced by inoculating the mucous membrane of the vulva of guinea-pigs, and in various other ways.

The disease, both in the human subject and in animals, is a local one; that is to say, the bacillus is found chiefly at the seat of inoculation, and does not multiply in the other tissues and organs of the body. In many cases the bacillus has also been found in small numbers in the lymphatic glands and in other organs, but this does not invalidate the view of the local nature of the disease.

The constitutional symptoms are caused by the absorption of toxines produced by the bacillus. These toxines are also produced in artificial cultivations; and, when separated from the bacilli by filtration and injected subcutaneously in sufficient quantity into animals, give rise to paralysis and death. Fatty degeneration of the organs and a peripheral nerve-degeneration are found post-mortem.

Although the bacillus is certainly the cause of the

disease, yet it may exist in the mouths of healthy individuals without producing any ill effect. Many instances have been recorded where the bacillus in a living and virulent state has been found in the mouths of healthy persons. In some of these cases the individuals have shortly afterwards developed diphtheria, but in others no departure from health has followed. The production of diphtheria depends upon the existence of a predisposition to the disease, in addition to the presence of the bacilli. We are at present unable to say what are the exact conditions which determine this predisposition. A lowering of the resistance of the tissues by the action of other bacteria may be one of the determining conditions. In any case the presence of virulent bacilli in the mouths of healthy individuals gives us some clue as to the origin and spread of the disease.

The diphtheria bacillus, like other pathogenic micro-organisms, varies in its virulence, and non-pathogenic varieties are met with. This variation is not necessarily associated with any morphological distinction; nevertheless the cases in which the "medium" variety are alone found are usually of a mild type.

The relationship of Hoffman's bacillus to the true diphtheria bacillus is of great interest. It is sometimes found associated with the diphtheria bacillus in cases of diphtheria. In addition it is often found in healthy individuals and in various inflammatory conditions of the throat, which from their history and clinical features appear to bear no connection with diphtheria. The bacillus is non-pathogenic to animals; and by no methods of cultivation has it hitherto been converted into the diphtheria bacillus, nor has the diphtheria bacillus been converted into it. In our experience no cases in which

Hoffman's bacillus alone was present have given rise to diphtheria in other patients. There does not appear to be any satisfactory evidence that this bacillus plays any part in the production of diphtheria. We must, however, state that some observers consider Hoffman's bacillus to be a non-virulent variety of the diphtheria bacillus, but that, under certain circumstances, it may acquire pathogenic properties.

The great similarity of the xerosis bacillus to the long variety of the diphtheria bacillus suggests that it may have some etiological connection with the latter. It has not yet, however, been rendered pathogenic to animals.

Various bacteria may be found in the diphtherial exudation producing putrefactive changes. The absorption of the products of putrefaction increases the severity of the disease. Still more important symptoms may be produced by a secondary infection of the fauces with streptococci. This may give rise to secondary suppuration in the lymphatic glands; and in association with the diphtheria bacillus is the cause of the lobular pneumonia which so frequently complicates laryngeal diphtheria. It has also been stated that the presence of streptococci in the throat increases the virulence of the diphtheria bacillus, and that the most severe cases of diphtheria are those in which many streptococci develop in cultivations made from the throat. Our own experience is opposed to this latter view, for we have found that the most severe cases of diphtheria are those in which almost pure cultivations of the diphtheria bacillus are found in the culture tubes. It appears to us that the dangers caused by the streptococcus are chiefly those of the secondary complications above mentioned.

The pathology of diphtherial paralysis is of interest

on account of the late period at which it may occur, after the membrane has disappeared and the other symptoms have subsided. It is analogous in this respect to the nephritis of scarlet fever. The primary symptoms of diphtheria are due to the action of the toxins upon the tissues, and the late paralysis can be explained in the same way. In the interval between the subsidence of the primary symptoms and the onset of the paralysis the toxins must be locked up and remain latent in the organs. We know that this is the case with certain pathogenic micro-organisms such as the malaria parasite, and Dr. Sidney Martin has found toxins in the spleen in diphtheria. His view is that the diphtheria bacillus secretes a ferment which converts the tissue proteids into albumoses, and that these latter bodies are the true diphtherial poisons.

Diagnosis.—*Faucial diphtheria* has to be distinguished from :—

1. The various forms of angina.
2. Scarlet fever.
3. Thrush.
4. Syphilis.
5. Tuberculous ulceration of the fauces.
6. Gangrene of the fauces.

1. It must be observed at the outset that certain very mild cases of diphtheria cannot be distinguished by clinical observation from *simple inflammation and ulceration of the tonsils*. We have already stated, and we must again draw attention to the fact, that diphtheria may occur without the formation of membrane. Indeed, in some cases there is no exudation whatever. Such cases are to be diagnosed only by means of a bacteriological examination, by a

history of contagion, or by a subsequent paralysis. Any definite enlargement of the lymphatic glands, the presence of albumen in the urine, or the fact that the disease is prevalent in the neighbourhood, should arouse suspicion ; and a bacteriological examination should be made to clinch the diagnosis. But when, to what appears to be a simple sore throat, is added croup, or a discharge, especially a blood-stained discharge, from the nose, the case is almost certainly one of diphtheria. On the other hand, it must be remembered that membranous exudation may result from other causes than diphtheria. We have already described such a condition as met with in scarlet fever. It may occur quite independently of this disease ; and such cases are to be distinguished from diphtheria only by bacteriological examinations.

From the severe, though uncommon, form of angina which goes under the name of *Erysipelas faucium*, diphtheria is more readily distinguished. In this affection there is intense brawny swelling of the fauces, high fever, and delirium, without, usually, the formation of membrane—a combination of symptoms that is uncommon in diphtheria.

Follicular tonsillitis may be mistaken for diphtheria ; and here again in some cases the diagnosis is by no means easy. In a typical example of this form of tonsillitis both tonsils are swollen, and the follicles are distended with exudation, which appears as yellow points upon the reddened surface. Such cases can readily be distinguished. But in some cases the exudation spreads from the yellow points over the adjoining surface of the tonsil, and this condition simulates diphtheria. There is little, if any, glandular enlargement, and no albuminuria. All we have said with respect to the diagnosis between

diphtheria and simple tonsillitis applies with equal force to these doubtful cases of follicular tonsillitis.

In *quinsy* there is much unilateral swelling of the tonsil and palate, with fever and severe pain. Pus quickly forms. In diphtheria the formation of pus is rare.

Another form of angina that requires notice is *herpes of the palate* and neighbouring parts. The herpetic vesicles soon rupture, leaving white spots, or small ulcers, surrounded by a red zone. Should there be many vesicles, a whitish patch is formed by their coalescence. There is very frequently also labial or facial herpes. Indeed, the existence of herpes about the lips or face will often give the observer a clue with respect to the nature of the angina; for it is only very occasionally that herpes is met with in diphtheria.

This is a convenient place to mention that *varicella gangrenosa* has been mistaken for diphtheria. The varicella pocks on the soft palate, becoming gangrenous resulted in unhealthy-looking ulcers, which were mistaken for membranous exudation. But both ordinary and gangrenous pocks were abundant upon the skin.

2. *Scarlet fever*. See p. 94.

3. *Thrush* may be found either in patches, or forming an unbroken sheet upon the soft palate. The deposit is whiter than diphtherial membrane, brushes off easily leaving a fairly normal mucous surface beneath, and is not attended by inflammation. The microscope reveals the presence of the *oidium albicans*. Thrush by itself gives rise to no constitutional symptoms; but it may arise in neglected cases of diseases such as phthisis, especially towards their termination.

4. *Syphilitic angina*.—The history, the presence of other syphilitic lesions, and in the tertiary stage the deepness

of the ulceration, together with the absence of membrane, are sufficient to distinguish syphilis. The error usually arises from mistaking the unhealthy base of the syphilitic ulcer for membrane.

5. In *lupus* and *tuberculous ulceration of the fauces* the history, the aspect of the lesion, the presence of lupus on the face, or the existence of tuberculous lesions elsewhere, are sufficient indications of the nature of the disease.

6. *Gangrene of the fauces* is not common in diphtheria. When it does occur it succeeds to extensive membranous formation and much œdema, and the constitutional symptoms are severe. In idiopathic gangrene of the fauces there is rapid loss of tissue, with the formation of an unhealthy-looking ulcer having a red edge. The ulcer enlarges day by day. It would perhaps be better to call the condition one of phagedœna rather than gangrene. The smell is excessively foul. There is continuous pyrexia, rapid emaciation, and towards the end (for the cases almost invariably end in death) much prostration. But it is astonishing how slight are the constitutional symptoms during the first two or three days of the disease.

Before quitting the subject of the diagnosis of faucial diphtheria two warnings may be given. The first is, that membrane may be present upon the fauces and yet escape detection, from being situated entirely behind the back of the soft palate or its arches, or low down upon the wall of the pharynx. The second is that, in not a few cases, even when pronounced constitutional symptoms are present, no complaint is made of the throat affection. This especially happens in children. A golden rule is never to omit to examine the fauces of a sick child.

Diphtheria confined to the *nasal passages* is very difficult to detect if no shreds of membrane are discharged. This

form of the disease does not appear to be common. The constitutional symptoms are usually mild, and the cases are often prolonged. The condition most likely to simulate nasal diphtheria is ulceration or sloughing of adenoid growths in the posterior nares.

Laryngeal Diphtheria.—In children it is easy to recognise that the larynx is affected, by the symptoms of obstruction that immediately arise. But in adults the membranous formation may have extended even to the larger bronchial tubes before the observer has any idea that the respiratory passages are affected, the size of the larynx preventing obstruction except in cases where the membrane is very thick. In adults loss of voice with dyspnœa, however slight, should suggest that the larynx has been invaded.

Diagnosis of Croup.—When a case of croup presents itself the cause of the obstruction must first be decided. It may be due to :—

1. Simple laryngitis.
2. Membranous laryngitis.
3. Some swelling external to the larynx—*e.g.*, post-pharyngeal abscess, growth, or extremely enlarged tonsils.
4. A foreign body in the larynx.
5. Spasm of the adductors of the vocal cords, as in laryngismus stridulus.
6. Paralysis of the abductors of the cords.
7. Ulceration of the larynx, simple, tuberculous, or syphilitic.
8. A laryngeal growth.
9. Œdema glottidis.

The age of the patient is an important factor in the diagnosis. In children the diagnosis almost always lies

between a simple and a membranous laryngitis; that is, for all practical purposes, between a simple laryngitis and diphtheria. If there is exudation upon the fauces, or an ichorous discharge from the nose, the case is almost certainly one of diphtheria. But in the absence of any affection of these parts, and in the absence of expectoration of membrane, the diagnosis is often very difficult. Evidence of exposure to the contagion of diphtheria is an important point.

It must be borne in mind that an attack of measles may begin with croup. In such cases there is pyrexia and coryza. Later the rash appears, and with its outcome the symptoms due to the laryngeal obstruction often abate, or even disappear. Croup commencing with a high temperature should therefore raise a suspicion of measles. But the same combination of symptoms may occur at the onset of an attack of diphtheria.

Croup arising during the eruptive stage of measles and scarlet fever is nearly always due to simple laryngitis or to ulceration; but in the former disease, and in the convalescent stage of scarlet fever, it may be due to true diphtheria.

Careful inquiry should be made for the history of a *foreign body*, and the pharynx, and as far as possible the larynx, should be explored with the finger, which may lead to the detection of a foreign body, or a post-pharyngeal abscess.

Paralysis of the abductors of the vocal cords is usually met with in adults, and is often due to locomotor ataxia. But it may occur as a part of a diphtherial paralysis.

In *laryngeal ulceration* (tuberculous and syphilitic), *growth*, and *œdema of the glottis*, the history and accompanying clinical phenomena are usually sufficient for the

purposes of diagnosis. They are most commonly met with in adults.

Laryngismus stridulus is usually seen in children who are the subjects of rickets and under two years of age. The attacks are attended with convulsive movements of the face or limbs, and with cyanosis ; they recur, and often take place only at night. Between the attacks there is no difficulty in breathing, and the child appears to be quite well.

Diphtheria of the vulva is most likely to be mistaken for noma vulvæ or erysipelas. But in diphtheria, if any necrosis of the tissues takes place, it is very superficial. Nor has the inflammatory area the same well-defined edge as is to be found in erysipelas.

Wound Diphtheria may be readily mistaken for various other infective conditions, and may only be recognised should membrane form. The history of exposure to contagion should always arouse suspicion.

Diphtherial Paralysis, following a mild and unrecognised attack of diphtheria, may be mistaken for paralysis due to some other cause, especially cerebral tumour and locomotor ataxia. In diphtherial paralysis there is never headache, nor optic neuritis, nor atrophy of the discs ; while vomiting occurs only in connection with symptoms, usually very serious, of cardiac irregularity and failure. Moreover, in paralysis following diphtheria, the palate and ciliary muscles are very frequently affected.

Before quitting the question of diagnosis we would again emphasise the importance of making a bacteriological examination in all doubtful cases. Even in cases where there is nothing abnormal to be seen diphtheria bacilli may be found upon the fauces. Thus a bacteriological examination of the fauces will often enable a diagnosis to be made in cases of laryngeal diphtheria, or even during

the paralytic stage of the disease, when the local exudation has cleared off.

Prognosis.—Diphtheria is a very fatal disease. Of 11,598 patients admitted into the hospitals of the Metropolitan Asylums Board during the years 1888 to 1894, 3,516 died, a fatality of 30 per cent.

Age is an important factor influencing the fatality. In the above series of cases about half of the children under five years of age died. A reference to the table given on p. 113 will show that the younger the child the more likely is an attack of diphtheria to prove fatal.

No case of diphtheria should be regarded as trivial. A case of faucial diphtheria, at first mild, may become severe, or the larynx may be invaded and life thus imperilled. Occasionally, too, we see very serious symptoms of cardiac failure suddenly arise in cases that have in the first instance appeared to be benign, and severe paralysis also may follow such attacks. But bearing these facts in mind, it may be said that in faucial diphtheria the more extensive, thick, and persistent the exudation or membrane, the worse the case, and that the gravity is increased when the nasal fossæ are involved. Those cases, also, are severe, in which the exudation has a gelatinous appearance. Epistaxis and hæmorrhage in other parts are unfavourable symptoms. Repeated vomiting is in all cases a very bad sign; so, too, though to a less degree, is pallor or a yellowish, waxy appearance of the skin.

Since death is often due to cardiac failure, and may take place quite suddenly, particular attention should be paid to the state of the pulse. Much caution is to be exercised in all cases where the pulse rate remains abnormally frequent or infrequent, even though the condition of the

fauces has shown great improvement. But often the onset of the symptoms of cardiac failure is gradual, and the patient slowly sinks.

Convulsions are of the gravest import, for they nearly always indicate that death is approaching.

The presence of much albumen in the urine is unfavourable. If with albuminuria the urine becomes scanty, and the pulse weak and abnormal in its rhythm, and more especially if there be also vomiting, the prognosis is exceedingly grave. It should be remembered that these symptoms may set in after all traces of exudation have disappeared from the fauces. As a rule, they occur about the fifth to seventh day of the disease. In nasal diphtheria, without affection of the fauces, the disease usually runs a benign course.

With respect to *complications*, the occurrence of croup is most unfavourable, and the more so the more insidiously the symptoms appear. For in such cases, by the time dyspnœa has become distressing, the disease has progressed along the respiratory tract further than has been suspected. This subject will also be dealt with under the head of Tracheotomy. Signs of invasion of the respiratory passages are of more serious import in children under two years of age, and in patients over fifteen, than in those of intermediate years.

Diphtherial paralysis is not a trifling complication. The mortality of the cases that have been observed at the Eastern Hospital was about 13 per cent. There is no relation between the severity of the paralysis and the length of time intervening between the attack of diphtheria and the onset of the paralysis. The more widespread the paralysis the more likely is the case to prove fatal. Usually the implication of the various groups of muscles

takes place quickly in these fatal cases. The prognosis becomes worse when the respiratory muscles are involved, whatever may have been the previous extent of the paralysis. The occurrence of syncope, vomiting, irregular respiratory rhythm, and a frequent or irregular pulse, are exceedingly unfavourable symptoms. If albuminuria persists during the attack of paralysis the prognosis should be guarded; even if unfavourable symptoms do not arise the duration of the paralysis may be long. Few cases, however, are prolonged beyond three months. It is excessively rare for any permanent paralysis to be left behind. Death during paralysis is usually due to cardiac failure, or to paralysis of the respiratory muscles. It may also be due to an attack of convulsions, or to suffocation from food getting into the trachea, or to septic lobular pneumonia from the same cause.

Cervical cellulitis and lobular pneumonia are grave complications.

Treatment.—*Local.* In faucial diphtheria the same local treatment should be employed as in scarlet fever (*vide supra*, p. 101), and it must be kept up for some days after the disappearance of the exudation, which is very likely to recur. Remembering that diphtheria is primarily a local disease, every effort should be made to treat the local condition on antiseptic principles.

Besides the remedies mentioned in the chapter on scarlet fever, the compound alkaline lotion of the Guy's Pharmacopœia will be found useful as a solvent of the local exudation. The formula is as follows:—

Bicarbonate of sodium	7 grains.
Borax	7 "
Common salt	7 "
Refined sugar	15 "

Mix for one powder, and dissolve in two fluid ounces of warm water.

We cannot recommend the application of strong caustics, such as hydrochloric acid or nitrate of silver. If it can be accomplished without setting up bleeding the membrane may be mechanically removed with forceps.

General.—On account of the liability to cardiac failure the patient should be made to keep the recumbent position as much as possible, and should for the same reason be kept in bed for some time after the exudation has disappeared, and all constitutional symptoms have passed off. A more stimulating diet may be allowed than is advisable in scarlet fever, and in addition to milk, beef tea, or the various patent extracts of meat may be given during the acute stage. Peptonised foods are also useful. In the case of young children it is often necessary to feed by means of an œsophageal tube passed through the nose, in consequence of absolute refusal to take nourishment. Solids may be given as soon as the condition of the throat allows. Alcohol is useful in certain of the cases in which symptoms of cardiac failure appear. Strychnine does good. It should be administered from the very commencement of the illness, either by the mouth in the form of liquor strychninæ, two to five minims every four hours, or hypodermically in doses of $\frac{1}{100}$ grain at the same intervals. Some form of iron may also be given with advantage, especially during convalescence.

Frequent vomiting is an exceedingly difficult symptom to allay. Peptonised milk or peptonised foods will occasionally be found useful, so also will nutrient enemata, or suppositories. Drugs are of little use.

In cases where the heart's action is frequent and irregular, and especially during the convalescent stage of the disease, strychnine and iron may be given with advantage. Digitalis is of no use. As long as

the irregularity continues the patient should be kept in bed.

Nasal diphtheria is treated by frequently syringing or flushing out the nasal passages with an antiseptic solution, as in the case of scarlet fever. Epistaxis should be met by the injection of iced water, or the external application of ice. Should these means fail, recourse may be had to the injection of pure oil of turpentine.

Diphtheria of the *vulva*, *conjunctivæ*, and *wounds*, is best treated by frequent washings with antiseptic solutions; while hot boracic fomentations should be applied in the intervals.

In *paralysis* iron and strychnine should be administered. Galvanism is also advantageous, but should not be employed for a week or ten days after the onset of the paralysis. Paralytic cases should always be kept in bed at the commencement. If, after two or three weeks, the paralysis remains limited to the palate or ciliary muscles, and no further development takes place, the patient may be allowed up again; but he should not be permitted to over-exert himself. The slightest sign of difficulty in swallowing, or of the entrance of food, solid or liquid, into the larynx, is an indication for feeding by means of the œsophageal tube.

The treatment of *laryngeal diphtheria* calls for special notice. Symptoms of croup will often be relieved, and operative measures be avoided, by placing the patient in a tent, in which the air is kept moist and warm by means of steam generated in an ordinary bronchitis kettle. A hot bath will at times cut short an attack of laryngeal spasm. Emetics are not only useless, but sometimes harmful.

The question, however, of operative interference is in

many cases certain to arise sooner or later. Two methods of relieving the obstruction are advocated, Intubation and Tracheotomy. Intubation is not very successful in obstruction due to membrane, and it is more difficult of performance than tracheotomy. In many cases, too, when it has been successfully accomplished, the relief given has been but temporary, and recourse to tracheotomy has been ultimately necessary. The latter operation is therefore preferable to the former.

The moment to be chosen for operative interference in croup complicating diphtheria varies much in different cases, and under different circumstances. If the dyspnœa is urgent, the patient restless, and seriously distressed, and there is marked recession, the operation should be performed at once. But there are cases in which, although the larynx is clearly involved, and even membrane may be coughed up, yet the dyspnœa does not at any time become urgent. Such symptoms of laryngeal implication may continue for some days, and the patient ultimately recovers without operative interference. It would be a mistake in hospital practice to operate in such cases, for it must be borne in mind that tracheotomy has certain risks of its own, both immediate and remote. We say in hospital practice, because there everything is ready should occasion arise, both operator and instruments. But in private practice it is a different matter. Slight symptoms are liable at any moment to become severe ; and when the medical attendant may have to be fetched from some distance it is not advisable to wait till urgent symptoms arise, but to decide upon operating as soon as the larynx shows signs of becoming seriously affected. Briefly, therefore, it may be said that, in many cases in hospital practice, delay is preferable to immediate

operation, but that this rule does not hold in private practice.

In that variety of the disease in which there are attacks of laryngeal dyspnœa, with intervals of freedom from distress, it is not advisable to let the patient have more than three such attacks ; and if the first or second attack is exhausting it is not advisable to wait for a second or third. In those cases in which the laryngeal symptoms gradually develop, tracheotomy should be performed before the obstruction becomes urgent, if the dyspnœa is sufficient to prevent the patient from getting rest.

In all cases of tracheotomy it is desirable to have the service of skilled nurses, in order to carry out efficiently the all-important after treatment.

In a work such as this a detailed account of the operation of tracheotomy would be out of place. Suffice, therefore, to say, that chloroform should be administered if there be time, that, as far as possible, hurry should be avoided, that the surface of the trachea should be well seen before the opening in it is made, and that a sufficiently large opening should be made before attempting to insert either forceps or tube. When the trachea is well opened, a pair of Bryant's forceps should be inserted, and no attempt should be made to put in the tube until the patient is breathing easily. Any membrane that can be reached should be removed from the larynx and trachea by means of forceps. Should there be any risk of blood getting into the trachea the patient should be inverted, preferably by tilting up the operating table, the trachea meanwhile being held open with forceps. Parker's tube is the best form to use. It is advisable to have a hot brandy or port-wine enema ready at hand, to administer immediately after the operation, if necessary.

The after treatment is of the greatest importance. Unless the ward is imperfectly warmed the patient does better without a tent. Every effort should be made to keep the wound aseptic. At the same time it should be disturbed as little as possible. Therefore (unless special circumstances, such as recurrence of dyspnoea, or hæmorrhage, occur) the tube and dressing should not be changed more frequently than once every twenty-four hours. By placing over the top layer of the dressing, immediately beneath the shield of the tube, a layer of gutta-percha tissue or jaconet, the dressings are prevented from becoming moistened and befouled by any mucus, etc., that may be coughed up. Two or three layers of gauze upon which have been sprinkled a few drops of menthol dissolved in olive oil (one in seven), should be lightly fastened over the opening of the tube. The tube should be left out as soon as possible, and should not be replaced unless it is necessary, which, however, is often the case for the first few nights after removal. If the patient refuses food, or if liquids come through the tracheotomy tube or wound, nutriment should be administered by means of an india-rubber nasal tube. The patient is better without sprays, except in those cases where no mucus comes up through the tube, and he suffers from a frequent and harassing cough, when a spray of bicarbonate of sodium (fifteen grains to an ounce of water) gives relief.

It is necessary to have a nurse who can be trusted to put the tube back if accidentally coughed out, or, at any rate, is able to hold the trachea open with a pair of Bryant's forceps till the medical man arrives. She should also be able to feed the patient by the nasal tube.

When the tube is left out the child should be encouraged

to breathe through its larynx. This may be accomplished by allowing it to blow bubbles with a pipe and soap-lather, or to blow a whistle or toy-trumpet. In some cases, even after three or four weeks, the tube cannot be left out for any length of time. Intubation will effect a cure unless the cause of the continued obstruction be due to granulations in the trachea around the tracheotomy wound. If this condition be suspected it is advisable to open up the wound and scrape away the granulations. Occasionally inability to breathe without the tube is due to nervousness, which may be overcome by the use of a blind tube that does not reach the trachea. Cellulitis around the wound is best treated by the frequent applications of hot boracic fomentations.

In certain uncommon cases of *abductor paralysis* (arising usually in the course of a generalised paralysis) intubation is of great value.

The fauces should be regularly inspected every three or four days during convalescence, in order to be certain that no recurrence of the disease is taking place.

The antitoxine treatment of Diphtheria. — Immunity against infection with diphtheria can be conferred upon animals in various ways. We owe to Behring and Kitasato the discovery that the blood serum of animals immunised to diphtheria possesses the property of protecting other animals from the effects of inoculation with virulent cultivations or of injection with otherwise fatal doses of the toxins. The substances contained in the serum capable of protecting against the toxins are called antitoxines, and it is customary to speak of the serum itself as "antitoxine" or "antitoxic serum."

The therapeutic properties of the antitoxic serum are well illustrated by the following experiments of Roux.

Guinea-pigs were inoculated with the diphtheria bacillus in the vulva, and typical false membranes were produced. Some of the animals were treated with antitoxic serum, with the result that the membrane cleared off and recovery ensued, while the animals which were not treated died.

This method of treatment is now applied in the case of the human subject. The antitoxine used is the blood serum of horses that have been immunised by repeated injections of the diphtheria toxine or of cultivations of the diphtheria bacillus. The toxine consists of a broth cultivation freed from bacilli by filtration, and is of such a potency that $\frac{1}{10}$ cc. will kill a guinea-pig weighing 300 grammes within thirty-six hours. The strength of the antitoxine is estimated by ascertaining the smallest amount which will neutralise ten times the fatal dose of the toxine when injected at the same time into a guinea-pig. A "normal unit" is ten times the amount of serum which is required to neutralise ten fatal doses of the toxine. A good antitoxic serum should contain as much as 100 normal units in each cubic centimetre.

The dose of antitoxine for the human subject depends partly upon the strength of the serum and partly upon the severity of the attack of diphtheria. Instructions as to dosage are usually given by those who prepare the serum. With serum of such a strength as has been mentioned the dose is 20 cc. to 40 cc. In a severe case this may be repeated in twelve hours, and may be continued daily until improvement ensues. The treatment should be continued until the exudation has disappeared and the constitutional symptoms have subsided.

The serum is administered by injecting it subcutaneously into the flank by means of a syringe, which is so

constructed that it can be sterilised by boiling. The needle of the syringe should be connected to the barrel by a piece of indiarubber tubing, to prevent injury in case the patient moves. The syringe, needle, and tubing should be boiled immediately before use, and the skin at the seat of injection should be washed with some antiseptic solution. If these precautions are taken, and the serum is pure, there is no risk of an abscess forming at the seat of injection, nor of any septic trouble occurring.

Under this treatment the exudation clears off more rapidly than with any other mode of treatment, the pulse improves, and the other constitutional symptoms subside. The effect upon the local affection is well shown in laryngeal cases, where the necessity of performing tracheotomy is frequently avoided. Moreover, in cases of faucial diphtheria treated by this method, laryngeal symptoms less frequently supervene. The value of the remedy has been shown by a large number of statistics comparing the mortality of cases treated with similar cases not treated. As an example, we will quote a series of cases under our own observation, in which it will be seen that the mortality sank under treatment to about half the average.

Case-mortality of diphtheria in children under fifteen at the Eastern Hospital (Goodall, Card, and Washbourn):—

		Cases.	Deaths.	Mortality per cent.
Not treated with serum.	{ 1893	397	166	41·8
	{ 1894 (Jan. 1—Oct. 22). . .	400	144	36
	{ Jan. 1, 1893—Oct. 22, 1894. .	797	310	38·8
	{ Sept. 14—Oct. 22, 1894 (39 days)	72	28	38·8
Treated with serum.	{ Oct. 23—Nov. 27, 1894 (36 days)	72	15	20·8

It is very important to commence the treatment immediately the disease is recognised, for the earlier the

remedy is used the better the chance of recovery. In the above series of cases, of 32 which were treated during the first three days of the disease, only 2 died, while of 29 which were treated at a later period 11 died.

It is difficult to estimate the effect of antitoxine upon

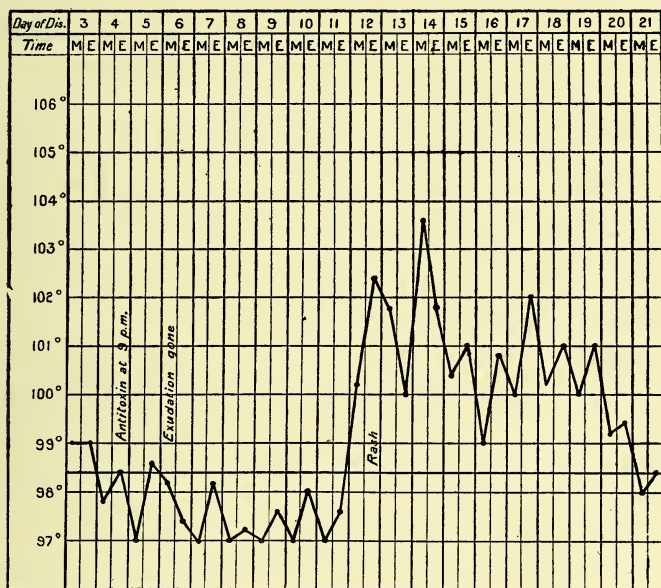


CHART G 2.

Slight case of diphtheria when first seen on the third day. Exudation increased from third to fourth day. Antitoxin 20 cc. at 9 p.m. on fourth day. Pyrexia seven and a half, and rash eight, days after the injection. The rash lasted six and a half days, the pyrexia nine.

the occurrence of paralysis for this reason: the more severe the attack of diphtheria the more likely is paralysis to ensue, and many cases fatal at an early stage would no doubt have developed paralysis had they recovered. Consequently, when the mortality is lowered by treatment, the actual number of cases of paralysis may increase, from the

survival of severe cases which would otherwise have died before paralysis had supervened. The comparison of statistics of incidence of paralysis is thus vitiated.

Antitoxine has been used not only for treatment, but also as a prophylactic measure against diphtheria. Its use in this respect should be strictly limited to cases where the risk of infection has been very great; for the protection afforded is not of long duration, and, besides, there are certain *after-effects* which frequently follow the injection of antitoxine, and which cause discomfort and inconvenience, although they do not endanger life. These after effects occur from one to three weeks after the injection. They are the *rashes* described on p. 51, *pyrexia*, and *joint pains*.

Joint pains are less frequent than rashes, and the periarticular structures are more involved than the joints themselves, although effusion into the latter may occur. There is generally pyrexia accompanying the joint pains, and often considerable constitutional disturbance. The rashes may or may not be accompanied by pyrexia. Some specimens of antitoxine more frequently give rise to rashes and joint pains than others.

It has been stated that antitoxine has an injurious effect upon the kidneys, producing anuria and nephritis. But the authors have never observed nephritis to follow the injection of antitoxic serum, nor is anuria more common in the serum-treated than in the non-serum-treated cases.

In all cases of diphtheria the antitoxine treatment should be adopted, but the local and general measures previously recommended should at the same time be carried out.*

* For further information with regard to antitoxine *vide* Appendix.

CHAPTER VIII.

MEASLES. MORBILLI.

MEASLES is an acute specific fever, of which the characteristic symptoms are a blotchy eruption and catarrh of the respiratory passages.

Etiology.—The *geographical distribution* is very wide, for the disease is met with in all parts of the civilised world, in the form of local but frequently recurring epidemics.

Season.—If we judge by the number of recorded deaths, measles is especially prevalent in Great Britain during two periods of the year—namely, from November to January, and in May and June.

Age.—It is almost entirely a disease of children, and especially of children under five years of age. As one attack confers protection, and as comparatively few children escape, it is but rarely seen in adults. If, however, the disease be introduced into a community, the individuals of which are not protected by a previous attack, adults and children are equally affected.

During the last few years there has been a decided increase in the mortality from measles in this country.

Dissemination.—Measles is a highly infectious disease. It is spread chiefly in a direct manner by those suffering

from it, but there is reason to believe that the infection may be harboured in, and conveyed by, fomites. It would appear that the secretions and exhalations from the nasal, buccal, and respiratory mucous membranes are especially infectious. The contagion is not so persistent as that of some of the other infectious diseases; and it is most active during the prodromal and eruptive stages. There is no evidence to show that the infection is ever conveyed by the water or milk supply.

The **incubation period** is somewhat variable; it ranges from seven to eighteen, but is on an average fourteen days.

Clinical History.—The *period of invasion* is usually of four or five days' duration. Though in most cases it commences suddenly, in a few the symptoms develop gradually and insidiously. The earliest symptoms are coughing, sneezing, coryza, and lachrymation, attended with moderate pyrexia. Occasionally there is vomiting, diarrhœa, or shivering. These symptoms continue for a day or two, and the patient seems to be suffering only from a severe "cold." On the second or third day there often is a *remission* of all the symptoms; the temperature falls even to normal, and the cough, coryza, and lachrymation almost entirely disappear (see Chart H). So marked may this remission be that any suspicion as to the true nature of the illness may be lulled, and the patient be deemed to be convalescent. But on the evening of the second, or any time on the third or fourth day, the original symptoms recur with still greater intensity. In addition the face usually becomes puffy, and often presents an ill-defined, red blotchiness. A few hours later, that is to say on the third, fourth, or fifth day, but more often on the fourth, the characteristic eruption makes its appearance. The remission described

above may be very slight or entirely wanting, even when the prodromal period is of three or four days' duration. It does not occur when the rash appears as early as on the first or second day.

During the prodromal period a fugitive uniform erythematous rash is occasionally to be observed upon the trunk, which may lead to a mistaken diagnosis of scarlet fever.

The *eruption* of measles most frequently comes out first upon the face, but it is by no means unusual for it to begin on the neck, trunk, or buttocks. It commences as small, red, discrete spots, irregularly scattered, and fading on pressure. These spots quickly become larger, and many of them can be distinctly felt, even when the skin upon which they are situated is stretched by the fingers. These papules, at first circular and discrete, by their individual

enlargement as well as by coalescence with one another, presently give rise to crescentic, serpentine, or irregularly shaped blotches. At a still later stage extensive tracts of skin are involved in a smooth erythema, which is raised at the edges, and which may contain islets of unaffected skin. In some cases, however, the papules and blotches

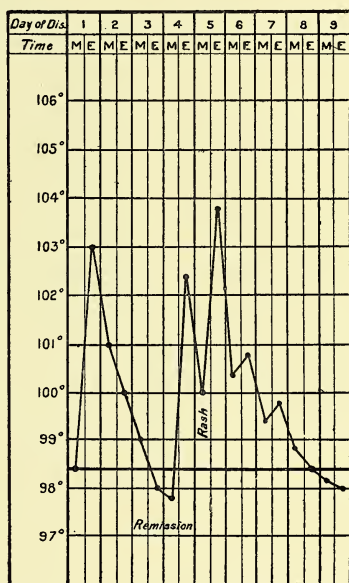


CHART H.

Boy, aged 2½. Mild case of measles, with well-marked remission. There was a prodromal rash on the first day. The child was fretful on the second, but seemed quite well on the third; catarrhal symptoms on the fourth; rash early on the fifth.

merge into one another, and a uniform erythema very like that of scarlet fever is produced. The eruption is



DIAGRAM IV.—MORBILLI.

A papular and macular rash affecting the whole of the skin.

at first of a pinkish colour, but as it comes out more fully the hue becomes a darker red, and often has a bluish tinge, especially if much bronchial catarrh is present.

It is not at all uncommon for it to become hæmorrhagic, even in moderately severe cases ; and then it does not fade



DIAGRAM V.—MORBILLI.

completely upon pressure. This condition is especially to be seen about the eyelids and ears and the flexures of the joints. In most cases the rash is universal, being seen from

the crown of the head to the soles of the feet. No region of the skin escapes; scalp, face (including the circum-oral ring), palms, and soles are all affected. Occasionally the eruption, though wide in distribution, is sparse. The face is always involved, even in those cases when the eruption has commenced elsewhere.

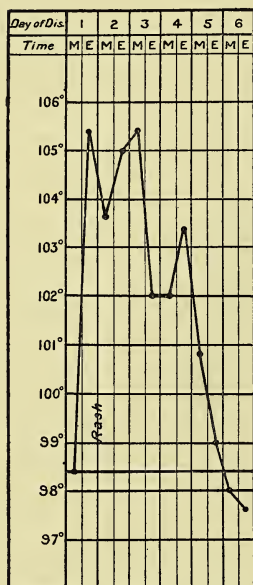


CHART I.

Girl, aged $2\frac{1}{2}$. Short, but sharp, attack of measles. Rash within twenty-four hours of onset.

The rash takes from twenty-four to seventy-two hours to attain its maximum development. It is common to see it fairly well marked on the face, but only slight elsewhere, for the first twenty-four or thirty-six hours; then suddenly it invades the other parts of the body.

Red blotches may also be observed in some cases on the mucous membrane of the mouth, palate, and pharynx.

The rash fades rather quickly, even when it has been hæmorrhagic. It first of all loses its papular character, and then the red hue disappears. A brownish staining is, however, frequently visible for some two or three weeks.

The rash may come out as early as the second or even the first day.

During the early stages of the eruption the *temperature* still continues to rise, and often reaches 104° Fahr., or more; 105° is not at all an uncommon temperature. But very soon after the eruption has attained its maximum development, and when it is just commencing to fade,

the temperature begins to fall, and drops to normal in from twenty-four to thirty-six hours—that is to say, by crisis.

With the appearance of the rash, and sometimes a day or two before, symptoms of *catarrh of the bronchial tubes*

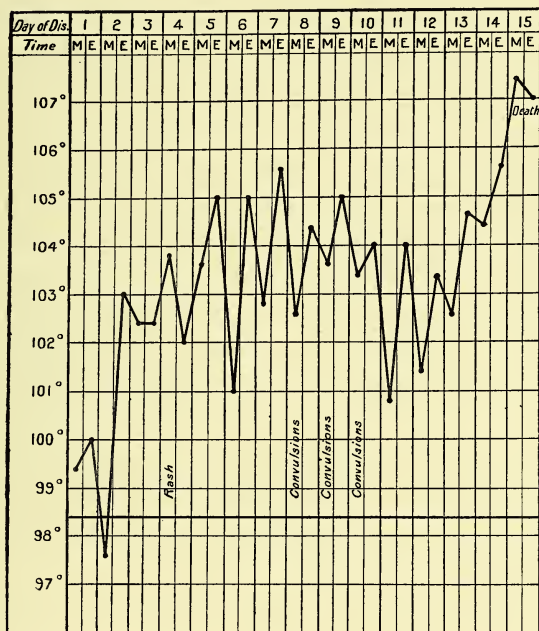


CHART J.

Girl, aged 2. Very severe case of measles, with much bronchitis; fatal at end of fifteenth day; convulsions on eighth, ninth, and tenth days.

set in, to become more pronounced during the next two or three days. The respirations are very frequent and short, and there is a frequent dry cough. Often the larynx is slightly involved in the catarrhal inflammation, as is shown by an alteration or loss of voice, and harshness of the cough. On physical examination the signs of bronchitis

are discoverable. A case with no bronchitis is exceptional, though in some instances it is very slight. In ordinary cases in which recovery takes place the bronchial inflammation clears up with the disappearance of the rash.

There is usually slight sore throat during the end of the prodromal and the beginning of the eruptive stage, with some swelling, and redness of the faucial mucous membrane. During the febrile stage the pulse rate is considerably accelerated, and is especially so when there is much bronchitis. The urine is concentrated, and may contain a trace of albumen. The skin is moist. The patient is restless, and sleeps only in short snatches. There may be delirium. The appetite is lost, but thirst may be a marked symptom. When the bronchitis is severe there is cyanosis, dilatation of the alæ nasi, and in young children slight recession of the lower portion of the thorax.

After the eruption has faded it is common to find a branny desquamation, which may continue for a week or ten days. In a few cases the desquamation is profuse, large flakes being detached.

The patient is usually well in ten days to a fortnight from the appearance of the rash.

In fatal cases the cause of death is nearly always bronchitis or lobular pneumonia. But death may be due to certain of the complications to be mentioned later.

Varieties—Writers describe, besides the ordinary form of measles, two others—viz., *morbilli sine catarrho*, and *morbilli sine morbillis*.

In the first of these two varieties a measly eruption and fever are present without the catarrhal symptoms (coryza, bronchitis, etc.). It is exceedingly doubtful, however,

whether such a form of morbilli exists ; for it is certain that rubeola (roetheln, or German measles), and blotchy eruptions due to other causes, have frequently been mistaken for morbilli.

With respect to *morbilli sine morbillis*, that is measles in which the febrile and catarrhal symptoms are present without the eruption, there are two classes of cases which come under this heading.

The one class, which is fortunately rarely met with, consists of cases fatal on the fourth and fifth day, before the characteristic eruption has developed. The proof that these are cases of measles rests on the history of exposure to contagion, and on the fact that they themselves have been the source of infection to others.

The other class of cases are mild forms of the disease, in which the catarrhal symptoms are slight, and the rash so ill developed as sometimes to escape detection.

Complications.—The most important are those of the *larynx* and *lungs*. As has been already stated, in nearly every case there is catarrh of the bronchial tubes to a greater or less degree ; and this may be so severe that the patient dies from bronchitis.

In other cases the parenchyma of the lungs may be involved, and a *lobular pneumonia* results. This is a serious complication, and is a frequent cause of death. Should recovery ensue convalescence is much retarded, and the patient remains ill for three to six weeks. Occasionally *lobar pneumonia* supervenes, and *pleurisy*, with or without effusion (usually purulent), may be met with.

It is not uncommon for *catarrh of the larynx* to occur, giving rise to harshness of the voice and cough, and even slight stridor. But the inflammation may be so intense as to cause laryngeal obstruction, with marked stridor, loss of

voice, metallic cough, recession of the thoracic walls, and cyanosis. Such symptoms usually come on during the eruptive stage, and (unless due to diphtheria) are rarely delayed till the period when the rash has faded. In a few cases an attack of measles commences with symptoms of laryngeal obstruction, which abate with the appearance of the rash.

Membranous inflammation of the fauces, larynx, trachea, and bronchial tubes is more common than is usually supposed. Sometimes only the larynx and the respiratory tract below it are affected, but the fauces may also be involved, though it is not common to find membrane upon the fauces alone. This complication, which, in the majority of cases, is *true diphtheria*, is exceedingly fatal. It usually supervenes during the eruptive stage, often just when the rash is fading; sometimes it arises later. The symptoms of laryngeal obstruction develop with great rapidity, and are accompanied by a rise of temperature.

Constant and obstinate *vomiting* and *diarrhœa*, especially the latter, may occur at any stage; but they are most frequently encountered when the rash is fading, and for a week or ten days after its disappearance. Fatal exhaustion may be induced, or a *marasmus*, from which recovery is slow. *Marasmus* also follows some cases of measles, in which there has been little or no disturbance of the alimentary tract.

The *eye* may suffer in several ways. Blepharitis and conjunctivitis are common. The cornea may be inflamed, with such results as opacities or ulceration. Occasionally a corneal ulcer will perforate, and acute panophthalmitis follows.

Otitis and *otorrhœa* are also common; and as a result

there may be a mastoid abscess, and even such grave events as meningitis, thrombosis of the cerebral sinuses, or pyæmia.

Cervical lymphadenitis, with or without suppuration, is occasionally met with; so also is *cervical cellulitis*.

Convulsions, limited or generalised, recurring at intervals of several hours, complicate only severe cases, and are of very serious import. Other nervous complications or sequelæ are very rare.

Rare, too, are *cancrem oris* and *noma vulvæ*; still more rare are *endo-* and *pericarditis*. It is not at all uncommon for an attack of measles to be closely followed by manifestations of *tuberculous disease*, usually of the lungs, but sometimes of the cerebral meninges or peritoneum. In most of these cases the mediastinal or mesenteric glands will be found in a caseous condition.

By some writers *whooping-cough* is said to be especially related to measles. But there is divergence of statement as to which of the two most frequently immediately precedes the other. Both are very common affections of childhood, and both are infectious. It is not therefore surprising that they are occasionally present at or about the same time in the same subject.

Relapses of measles are rare, and **second attacks** are uncommon.

Length of Infectivity.—Isolation should be enforced for three weeks from the appearance of the rash, or as long as any complication, such as lobular pneumonia, is present.

Morbid Anatomy.—There are no characteristic post-mortem appearances found after death from measles. In the majority of cases bronchitis and broncho-pneumonia are present. Pleurisy, diphtheria, caseating bronchial

or mesenteric glands, or a more generalised tuberculosis, may also be found.

Pathology.—Nothing definite is known about the cause of measles. It is doubtless a living micro-organism, but it has not yet been satisfactorily demonstrated.

Most of the complications are due to a secondary invasion by various micro-organisms. Diphtheria is caused by the diphtheria bacillus, and broncho-pneumonia generally by a streptococcus.

Diagnosis.—Morbilli has to be distinguished from *rubeola* (*roetheln*), *scarlet fever*, the prodromal rash and early stage of the characteristic eruption of *small-pox*, and *typhus fever*. The differential diagnosis is discussed in the chapters dealing with those disorders. It may be well to mention here, however, that in *rubeola* there are little, if any, catarrhal symptoms, slight coryza at the most; the eruption comes out on the first or second day; and the posterior cervical and mastoid glands, and sometimes those in other places, are enlarged. In *scarlet fever* the rash usually appears on the second day, and avoids the face; and catarrhal symptoms are wanting. In *typhus* again catarrh is absent; the rash avoids the face, and is never so definitely papular as that of measles.

The eruption of measles may also be confounded with *papular and macular erythemata*, whether idiopathic, attending septic conditions, or following the administration of certain drugs. It is not very uncommon to find in septic cases of scarlet fever a secondary papular rash, which usually appears first and is particularly well marked upon the extensor surfaces of the extremities, though it may affect the face and trunk. No other symptoms simulating measles are, however, present. The same point is to

be observed with respect to the other forms of erythemata mentioned.

Prognosis.—The case-mortality (fatality) varies widely according to the character of the epidemic. It has been known to be as low as 2 per cent. in one epidemic, and as high as 40 per cent. in another. Age has a considerable influence on the mortality, the rate being high at all ages under five, and highest in the second year of life. Under two years of age the rate of mortality is greater in males, above two years in females. These statements are based upon the registered deaths.

The course of the disease is much affected by the nature of the circumstances in which the patient lives, the children of the poorer classes more readily falling victims than those of the wealthy. Bad ventilation, want of warmth, and lack of suitable nourishment, are the chief causes of this difference.

Measles is chiefly fatal by reason of the inflammation of the respiratory tract. Therefore, the more severe this is, the more grave is the prognosis. If the temperature does not fall with the fading of the eruption there is almost always a persistence of the pulmonary inflammation. (See Chart J.)

Implication of the larynx is especially serious ; so also are convulsions. Diphtheria is the most dangerous complication, for the larynx is nearly always invaded, and the membranous inflammation rapidly spreads to the bronchial tubes.

Vomiting and diarrhœa are also serious symptoms, especially the latter, particularly when it occurs in a very young patient. Marasmus following an attack of measles should give rise to suspicion of latent-tuberculous disease.

Treatment.—The treatment of measles resolves itself

chiefly into the treatment of bronchitis or other lung affections, or of any complication that may arise. It is very important to see that the air of the room or ward in which the patient is placed be kept at a temperature of not less than 60° Fahr. The patient should be protected from draughts by a screen or a tent; the air, should not be allowed to become too dry, as is often the case when the ward is warmed by hot-water or steam pipes. Under such conditions the ordinary bronchitis kettle is of use. If circumstances prevent the air of the room being kept up to the temperature above mentioned, the child should be kept in a cotton-wool jacket, which is preferable to the use of poultices to the chest. Of drugs, carbonate of ammonia, ipecacuanha wine, and tincture of squills, are efficacious. In cases in which there is much restlessness compound tincture of camphor may be given in combination with these drugs. In severe cases alcohol may be given in moderate amount with advantage; it is best given as brandy or port wine.

The diet should consist of milk and beef tea or chicken broth during the febrile period. As soon as this has passed bread and butter, fish, and eggs may be allowed, and after a few days ordinary diet. If the child absolutely refuses its food recourse must be had to the œsophageal or nasal tube.

If there is laryngitis and laryngeal obstruction intubation or tracheotomy may have to be performed. Operative measures should not, however, be undertaken unless the dyspnœa is urgent; but if it is certain that relief is required on account of the laryngeal obstruction, the presence of bronchitis or of pneumonia is no contra-indication to operative measures, whatever influence it may have upon the ultimate prognosis.

Vomiting sometimes requires treatment. A mixture containing some preparation of bismuth, or the use of peptonised food, will often check it. Diarrhœa more often requires attention than vomiting; it is a very troublesome complication, and at times difficult to stop. The use of peptonised food, or of cream, with small doses of brandy, should be first tried. The following mixture (which is taken from the Guy's Hospital Pharmacopœia of 1891) will also be found useful:—

Carbonate of bismuth	2 grains.
Aromatic chalk powder with opium	1 grain.
Glycerine of tannic acid	5 minims.
Mucilage of acacia	15 minims.
Water to a drachm.					

Or a mixture containing five to ten minims of dilute sulphuric acid and one or two minims of laudanum.

If these means fail, compresses of cold water, or even of ice, applied to the abdomen for a few hours, as recommended by Dr. Goodhart, are certainly worthy of trial, provided the patient be not collapsed. Raw beef juice will also at times be found very efficacious in these cases of vomiting and diarrhœa.

It is not often necessary to interfere with the temperature; but if there be any reason to suppose that a high temperature is having a bad effect upon the patient, the best treatment is a bath of about 98° Fahr., or the wet pack with the sheets wrung out of tepid water.

The greatest care should be taken that any discharge from the eyes is removed frequently, by bathing with a solution of boracic acid or permanganate of potash.

Other complications should be treated on ordinary principles. If diphtheria complicates the case the treat-

ment should be the same as for that disease ; but the prognosis is extremely unfavourable.

During convalescence, when there is wasting, cream, cod-liver oil, and some preparation of iron should be given.

The patient may be allowed out of doors at the end of three or four weeks if the weather be favourable.

CHAPTER IX.

RUBEOLA. ROETHELN. GERMAN MEASLES.

RUBEOLA is a specific fever attended by slight constitutional symptoms, and characterised by the early appearance of a papular or macular eruption. Complications do not occur, and the disease always terminates in recovery.

Etiology.—There has been much discussion concerning the nature of this complaint. Some writers have considered it to be a hybrid between scarlet fever and measles; others have thought it to be a modification of measles; others again have regarded it as a distinct disease having no connection with either measles or scarlet fever. The latter is the view which we adopt, though it is certainly sometimes very difficult to distinguish rubeola from measles, and in a certain stage from scarlet fever.

The main proof of the specific nature of the disease lies in the fact that it does not protect against either measles or scarlet fever, nor do either of these diseases protect against it. As instances of this statement we give the following facts:—

A boy suffering from scarlet fever was admitted into the London Fever Hospital. He had come from a school where scarlet fever and rubeola were epidemic.

Shortly after admission he developed rubeola, which he had undoubtedly contracted at the school. His case



DIAGRAM VI.—RUBEOLA.

A papular, and at times macular, rash affecting the whole of the skin. Usually less intense than that of morbilli. The rash often becomes scarlatiniform on the trunk and extremities.

gave rise to an outbreak of rubeola among the scarlet fever patients in the ward.

At the Eastern Fever Hospital, during the summer of 1892, first rubeola, and shortly afterwards morbilli, broke

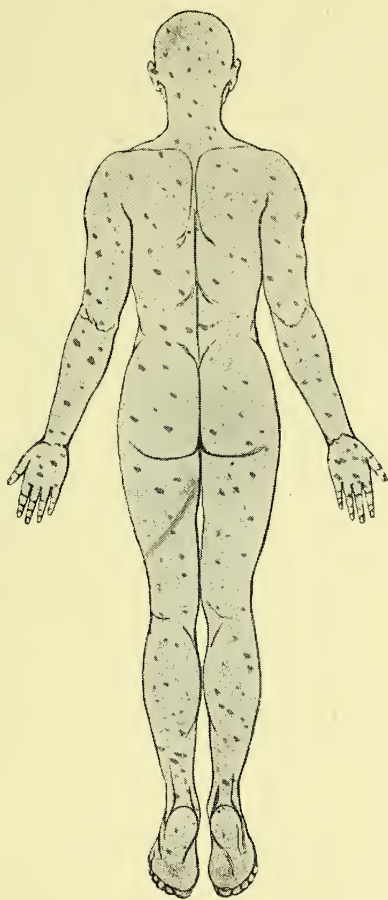


DIAGRAM VII.—RUBEOLA.

out among the patients in one of the scarlet fever wards ; and four children, convalescent from scarlet fever, underwent successive attacks of rubeola and morbilli.

Geographical distribution and seasonal prevalence.—

Rubeola occurs in epidemics in America, India, Egypt, the Continent, and Great Britain. In the latter country it appears to be more prevalent between March and June than at any other season of the year.

The *mode of infection* is usually by direct contact of the sick with the healthy. It is rare for it to be spread by means of fomites or third persons. The disease is very infectious, but perhaps not quite so infectious as measles.

Age and sex.—It occurs at all ages up to thirty, but over that age is not often met with. It would appear to be more common among young adults than among children. Sex seems to have no influence upon its incidence.

The **incubation period** varies from eleven to eighteen days; it may be longer, but is rarely shorter.

Clinical History.—Sometimes the patient complains of malaise, slight coryza, or sore throat for a day or two before the rash is noticed, but as a rule the rash is the first symptom of the disease. The posterior cervical, occipital, and mastoid *glands* occasionally become enlarged a day or two before the appearance of the eruption—in a few cases this enlargement is noticed a week or so before—and all the superficial lymphatic glands may be enlarged.

The *rash* almost invariably commences first on the face, and rapidly spreads to the trunk and extremities; the circum-oral ring is involved, and frequently the palms and soles. It starts as small, very slightly raised papules, similar to those of measles. On the face the spots fade quickly, remain discrete till they disappear, and leave no stain. Sometimes the papules run through a similar course on the trunk and limbs, but they often enlarge and coalesce,

so as to produce a uniform erythema, which may be distinctly punctate; under these circumstances it is often quite indistinguishable from the rash of scarlet fever. The rash upon the trunk and limbs lasts two or three days, and disappears earlier than that of measles, but a faint brownish staining may be left behind for some days.

The *constitutional symptoms* are often very slight. In many cases the temperature remains normal, and the patient feels in ordinary health. But as a rule there is slight malaise, and the temperature is raised two or three degrees; and it is not uncommon to find it as high as 104° or 105° Fahr. A point of importance is that even with so high a temperature as 104° or 105° Fahr., the patient does not appear to be seriously ill. The pulse-rate is moderately accelerated. The throat is usually dry and uncomfortable, and there is redness and slight swelling of the fauces. There is often a little conjunctivitis and nasal catarrh. Enlargement and tenderness of the lymphatic glands have already been mentioned as of early occurrence. The swelling is usually moderate; suppuration never takes place. A slight and transient albuminuria occurs in a small proportion of the cases. In two or three days the temperature falls to normal, the rash disappears, and the patient is convalescent. In some cases a fine branny desquamation follows the disappearance of the rash.

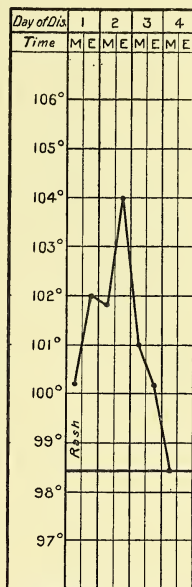


CHART K.

Female, aged 26. Well marked attack of rubeola; rash appeared on first day and lasted till the end of third.

There are no **complications** nor **sequelæ**. **Relapses** do not occur.

Protection.—One attack affords a fairly good protection, but second attacks certainly occur.

Length of Infectivity.—A patient may be considered free from infection ten days after the appearance of the rash.

Pathology.—Except that the disease must be due to a living micro-organism, we know nothing definite about its pathology.

Diagnosis.—Rubeola is most likely to be mistaken for measles, scarlet fever, and various drug rashes ; it may also be mistaken for the prodromal rash of small-pox.

In *measles* there are usually marked prodromal symptoms for two to four days before the appearance of the rash, these symptoms being pyrexia, cough, and coryza. In rubeola there are no prodromal symptoms in most cases, though occasionally slight coryza and enlargement of glands may be present. The rash of rubeola is of a brighter hue than that of measles. An important point of distinction is the mildness of the constitutional symptoms, even when the temperature is high. A patient suffering from measles is always distinctly ill when the temperature reaches 103° Fahr. ; but in rubeola a temperature of 104° Fahr. is often unaccompanied by marked constitutional disturbance. Bronchitis is common in measles, but absent in rubeola.

The cases of rubeola that are liable to be mistaken for *scarlet fever* are those in which the rash has disappeared from the face, and has reached the scarlatiniform stage upon the trunk and extremities. In these cases the history of the rash on the face, the enlargement perhaps of all the superficial glands, and the mildness of the throat affection and constitutional symptoms, will generally,

though not always, render a correct diagnosis possible. Neither the "peeling" nor the "strawberry" tongue are observed in rubeola; nor in this disorder is the desquamation so profuse or definite as that which follows an attack of scarlet fever. Circumoral pallor is wanting in rubeola.

The diagnosis from *drug-rashes* and other forms of *erythema* will be found in the chapter which treats of those subjects.

Prognosis.—The disease is always mild even if the temperature is high. Recovery always results, and there are no complications nor sequelæ.

Treatment.—The patient should be kept in bed until the temperature has fallen, and should be isolated for ten days from the appearance of the rash. The administration of drugs is unnecessary.

CHAPTER X.

VARIOLA. SMALL-POX.

SMALL-POX is an infectious disease accompanied by a characteristic eruption which, starting as papules, passes through a vesicular into a pustular stage. The prodromal and the eruptive periods are attended by pyrexia, which takes a characteristic course.

Etiology.—The disease is *disseminated* chiefly from patient to patient; but it may be conveyed by fomites, or by a third person. The virus may be carried through the air by the exhalations of patients suffering from the disease; and there is some evidence to show that the infection can be conveyed for a considerable distance around the neighbourhood of a hospital in this manner. Direct inoculation with the contents of the pocks will also produce the disease. It is stated that a patient is most infectious when the pocks are becoming pustular; but he is infectious during the prodromal stage, and during the whole of the eruptive stages, until the scabs have separated.

Geographical Distribution.—Small-pox has occurred in most parts of the world, though it has successfully been prevented from obtaining a footing in Australia, Tasmania, and New Zealand. All races are liable to be attacked, and negroes are said to be especially susceptible.

Seasonal Prevalence.—The disease has a preference for certain seasons of the year. In London the number of cases begins to rise towards the end of November, increases up to April and May, during which months the largest number of cases occur, and then falls rather rapidly during June. The smallest number of cases is found in September.

Age.—No age is exempt from the disease. Children have been born with the eruption of small-pox well developed. In unvaccinated communities children are those who chiefly suffer; but in a country like England, where most of the children are vaccinated, adults are more frequently affected; for the protection afforded by vaccination diminishes in proportion to the period that has elapsed since the operation was performed.

Certain individuals are insusceptible to infection. This was well recognised in the days when inoculation was in vogue as a preventive; for sometimes repeated inoculation proved unsuccessful.

Before the days of vaccination small-pox was prevalent to a much greater extent than at present, and was the cause of an enormous number of deaths. Its first appearance among unprotected populations has been attended by a terrible mortality. In Mexico it is stated to have rapidly destroyed between three and four million people, and whole tribes in the West Indies are said to have been completely exterminated.

Small-pox was very prevalent throughout England during the years 1870 to 1873, but since that period it has occurred in the form of localised epidemics, which, at different times, have broken out in various parts of the kingdom.

Incubation Period.—The incubation period of small-pox

is more constant than in most other infectious diseases. It is usually twelve days; but it may be a day or two more or less. When following direct inoculation it is from eight to ten days.

Clinical History.—The *prodromal stage* generally begins suddenly with a rigor, or with a succession of slighter chills; then follow headache, and pains in different parts of the body and limbs. Lumbar pain is especially common, and it may be so severe as to give rise to a diagnosis of lumbago. The temperature rises quickly to 102° or 103° , and towards the end of the prodromal stage it may reach 106° , even in cases which are subsequently mild. Vomiting and epigastric pain are frequently met with, and diarrhœa is common in children. Sore throat, with redness and slight swelling of the tonsils and palate, is not infrequent, and when this is accompanied by hoarseness, lachrymation, and sneezing, it may lead to a diagnosis of measles. The skin is usually dry. The patient may be delirious, and in grave cases collapse sets in. The pulse-rate is increased in proportion to the temperature. The respirations are also increased in frequency, and may be so laboured that dyspnœa may become a prominent symptom. In children convulsions sometimes occur. In women the menstrual flow may set in before the usual time.

During the prodromal period certain *initial rashes* are sometimes met with. The most characteristic is the “triangular rash,” which is limited to a triangular space with the base at about the level of the umbilicus and the apex at the pubes. It consists of minute, closely set petechiæ situated upon an erythematous skin. Less characteristic are various erythemata which may be either maculated or uniform. The distribution of these rashes

may be general, affecting the face, trunk, and limbs, or may be limited to the extensor surface of the limbs, especially in the neighbourhood of the elbows, knees, wrists, and ankles. The maculated form rarely affects the face. These erythemata may be accompanied by the characteristic "triangular" petechial eruption already described. A prodromal urticarial rash is sometimes met with, but is rare.

The length and the severity of the prodromal stage vary. Three days is the usual duration in a case of unmodified small-pox; but it may be longer, and in cases of modified small-pox it may be absent, the eruption being the first sign of the disease.

The *eruption* comes out in the large majority of cases on the third day of the disease. We will take the history of a single pock. It begins as a red spot hardly raised above the surface of the skin. This soon becomes papular, and can be easily felt as a firm round body, to which the term "shotty" has been applied. Sometimes it can be better felt than seen. The papule rapidly increases in size during the first twenty-four hours, and by the end of the second day of the eruption a minute vesicle, at first conical in shape, is formed. During the next two or three days the vesicle becomes larger, more globular in shape, and of an opaline appearance. There is now a central depression, or umbilicus, in most of the pocks, which are also loculated, as is shown by the fact that they do not collapse when pricked. By the sixth or seventh day of the eruption the vesicle becomes completely converted into a pustule. Meanwhile an inflammatory change has been taking place in the surrounding skin, and a red areola is formed around the pustule. After this maturation, as it is called, of the pustule has occurred,

the process of desiccation begins. Many of the pustules are accidentally ruptured, others rupture of themselves, and a thick, honey-like, offensive material oozes out. This dries up, and with the roof of the pock forms a scab. But the pustule often dries up, and forms a scab without previously rupturing. The scab is of a yellowish-brown colour, often reddish or black from an admixture with blood. If the cutis vera has not been severely affected by the inflammatory process, the scab falls off in four or five days (twelfth or thirteenth of the eruption); but if the cutis vera has been much involved the affected portion becomes necrosed, and the slough takes three or four days longer to separate. In the first instance only reddish-brown stains are left, which after some time disappear without scarring; but when the cutis vera has been severely affected a scarring more or less pronounced is the result. At first the scars are pinkish or reddish-brown in colour, but after a considerable time they become white.

In many cases of small-pox the period of scabbing is attended with intolerable itching. When the pocks are numerous the hair falls out during convalescence, and the patient is left more or less bald.

The eruption usually appears first upon the face and scalp, especially about the forehead and lips; very shortly after it is seen also on the wrists. Later on it appears upon the trunk and the remaining parts of the upper extremities, and lastly, upon the lower extremities. The soles and palms are affected. It takes about three days for the rash to come out. On account of the later appearance of the eruption on the lower extremities, it is not uncommon to find pustules on the face at a time when the rash on the legs is only in the vesicular stage.

Although the eruption, as a whole, invades the parts in the order mentioned, yet at times a few more pocks may make their appearance in parts that have already been affected. The face and scalp, back of the trunk, and the distal portions of the extremities, especially the upper, are usually more affected than other parts.

Considerable swelling of the skin of the face, hands, feet, and genitals may occur when the eruption is profuse.

During the later stages of the eruption large flat bullæ, like those of pemphigus, not infrequently appear, especially upon the limbs.

The pocks are seen in greater numbers where the skin may happen to have been recently irritated, and are sometimes observed to follow the line of a garter or a belt. It is uncommon to find a pock on the conjunctiva. The number of pocks varies immensely in different cases.

The outcome of the eruption is often accompanied by severe itching and consequent scratching. On the palms and soles the pocks cause severe pain in cases where there is much swelling.

The eruption also affects the *mucous membrane* of the pharynx, palate, mouth, and tongue ; in fact, it often appears early on the pharynx and palate. The larynx, trachea, and large bronchi may also be the seat of the eruption at a fairly early period. On a mucous membrane the pocks appear first as red spots or blotches, afterwards as greyish round spots surrounded by a red zone.

The process of development of the individual pocks has been already described, but there are one or two points which remain to be mentioned. Even in unmodified cases a few of the pocks may be seen which are ill developed, and which do not go beyond the stage of papules surmounted by small vesicles. It is not

uncommon to find hæmorrhages in the pocks, especially

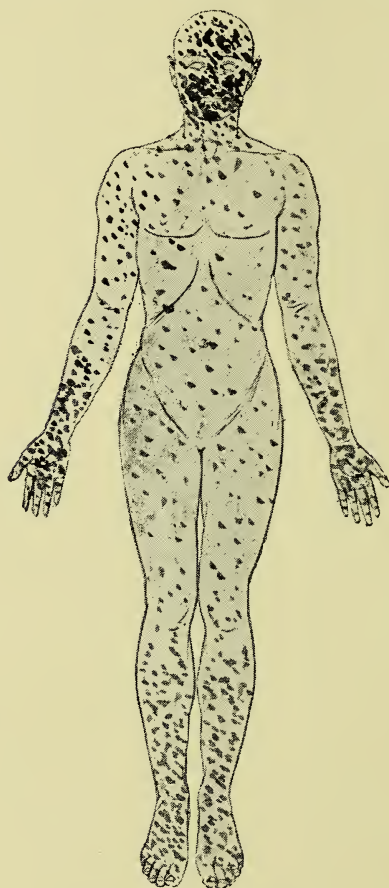


DIAGRAM VIII.—VARIOLA.

In variola, the eruption, which goes through certain stages (papules, vesicles, and pustules), affects the whole of the skin, but chiefly the face and head, the distal portions of the extremities, and the back of the trunk. In confluent cases the confluence is usually limited to the face and hands. In very mild and modified cases the distribution of the eruption is not so regular.

on the lower extremities; in some cases this is due to

injury caused by friction of the bed-clothes or of the legs against one another. It is important not to confound



DIAGRAM IX. —VAR.OLA.

these hæmorrhages with those found in hæmorrhagic small-pox. The hæmorrhage in the former cases occurs at a later stage; and after the sixth day of the eruption

extensive hæmorrhage may occur in the pustules without being of serious import.

As long as the pocks remain distinct from one another the disease is termed *discrete* small-pox. But in some cases the pocks are so numerous, especially upon the face and hands, as to leave hardly any healthy skin between

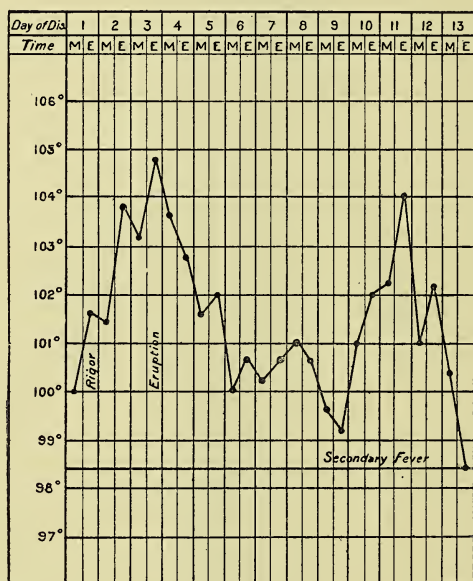


CHART L.

Discrete unmodified small-pox; recovery.

them; the disease is then termed *semi-confluent* or *coherent*. It is *confluent* when the pocks are so numerous in the regions mentioned that they cannot be distinguished from one another, having run together so as to form irregularly shaped figures.

The course of the disease depends upon the character of the eruption, whether it is discrete or confluent.

DISCRETE SMALL-POX.—With the appearance of the rash the temperature falls and becomes normal, or nearly normal, within two or three days. At the same time most or all of the symptoms subside, and the patient, except for the eruption, appears to have recovered. But when suppuration occurs in the pocks—that is to say, about the ninth or tenth day of the disease—a *secondary fever*, or *fever of maturation*, arises. The temperature goes up to 102° or 104° , the patient feels ill, complains of headache and sleeplessness, is restless, and may even be delirious. The pyrexia, which is of a remittent type, falls again after a few days. With the fall in the temperature the constitutional symptoms subside, and recovery quickly ensues.

SEMI-CONFLUENT SMALL-POX. — In semi-confluent small-pox the symptoms are considerably more marked. The temperature, even in these cases, often falls nearly or quite to normal, and the constitutional symptoms abate soon after the appearance of the papules. But, on the other hand, there may only be a remission of the pyrexia, and the patient may suffer from sleeplessness and delirium. The mucous membranes, often of the fauces and sometimes of the larynx, are affected with the eruption, and there is sometimes swelling of these parts leading to difficulty in swallowing, hoarseness, and symptoms of laryngeal obstruction. The saliva is secreted in excess, the tongue is occasionally much swollen, and various complications may arise. About the fifth day of the rash (seventh of the disease) the temperature rises again, and soon reaches 103° or 104° , or even more. The patient's condition grows worse, and delirium often appears, if it has not appeared before. The delirium is at first of an active nature, and in some cases the patient is extremely

violent and difficult to manage. In unfavourable cases, even if no complication occurs, prostration eventually sets in, the patient falls into the typhoid state, sordes collect upon the lips and gums, and there is muttering delirium and muscular tremor. The period of secondary fever is

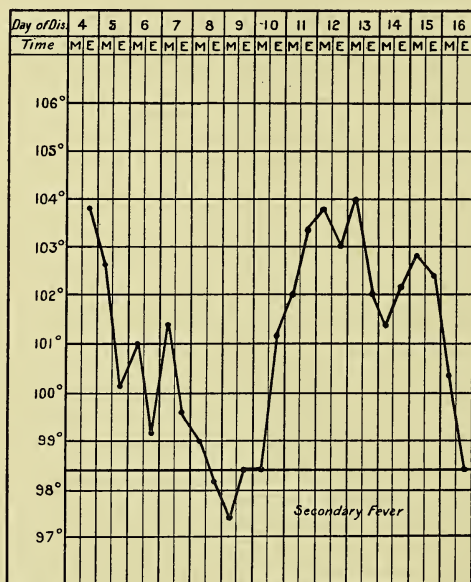


CHART M.

Confluent small-pox; recovery. The eruption appeared on the fourth day.

that in which death most frequently occurs. It is not often that patients die before the eleventh day, but many succumb on the twelfth to the fourteenth day of the disease. Immediately before death there may be hyperpyrexia.

In many cases, however, the symptoms are not so severe. The temperature may not rise above 103° , and

though sleeplessness is a common trouble, delirium may be almost or quite absent. About the eleventh, twelfth, or thirteenth day of the rash (thirteenth to fifteenth of the disease), the pyrexia abates, and the patient rapidly convalesces. In the more severe cases the fever is more

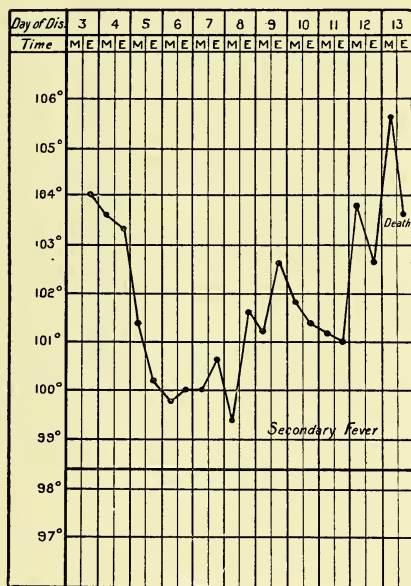


CHART N.

Fatal confluent small-pox. Eruption on third day.

lasting, and may not disappear till the end of the third week.

CONFLUENT SMALL-POX.—The eruption becomes confluent usually during the vesicular stage, although even the papules may be confluent. The skin may be much swollen, giving a bloated appearance to the face. The same condition is observed on the ears, neck, scalp, and

hands. The swollen state of the eyelids prevents the patient from opening his eyes. In many of the worst cases, however, there is little or no swelling of the skin. When the vesicular stage is reached the vesicles coalesce and quickly fill with a milky fluid; the face then looks as if it were covered with a mask, and the features are

quite unrecognisable. The skin, especially of the palms and soles, is very painful, and the pain becomes intense as sup-
puration ensues.

The constitutional symptoms are similar to those described in semi-confluent small-pox, but are more severe. The temperature usually falls considerably when the eruption appears, though in some cases the fall is slight. Delirium, and sometimes severe vomiting, are present during the eruptive stage, and all the symptoms increase during the secondary fever.

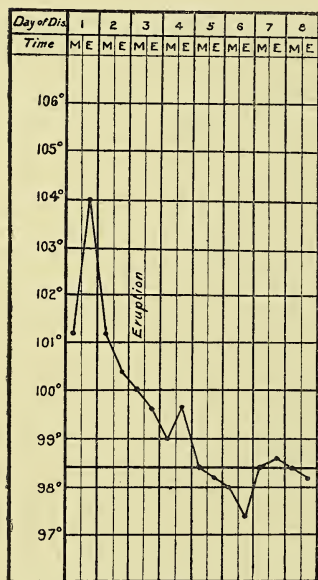


CHART O.

Mild discrete modified small-pox. Eruption on third day.

Some 60 or 70 per cent. of the cases die, death generally occurring on the twelfth, thirteenth, or fourteenth day of the disease.

ABORTIVE CASES. (*Modified small-pox, varioloid.*)

1. Occasionally, especially when the initial stage has been mild (though it may take place when the prodromal symptoms have been sharp), the symptoms subside without the eruption making its appearance. This form of the

disease is known as *variola sine variolis* or *variolous fever*.

2. The initial stage may be either mild or moderately severe, of short or of long duration, or even absent; and then the subsequent attack may be modified, especially

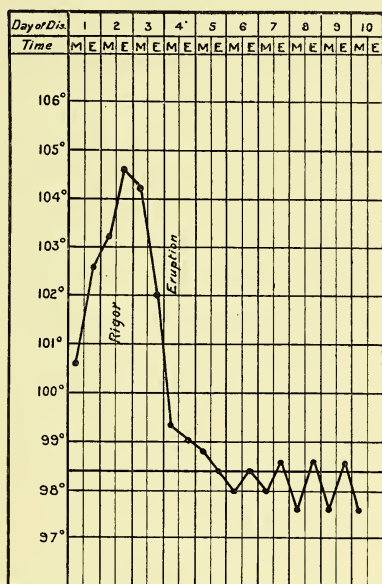


CHART P.

Mild discrete modified small-pox. Eruption on fourth day. Marked prodromal stage.

with respect to the appearance and course of the eruption, The eruption may be very scanty, or it may not have the usual distribution. It may not go beyond the papular or the vesicular stage, even though it is profuse; a few papules may become vesicular, or vesicles pustular, as the case may be, the remainder aborting. Or the vesicles and pustules may be very imperfectly developed. Lastly, the

individual pocks may change from papules to vesicles and pustules very much more quickly than in the unmodified form of the disease. In these cases the papules may be seen earlier on the trunk than on the face. When vesicles and pustules do form they are apt to dry up very quickly. As far as the constitutional symptoms are concerned the initial stage terminates very abruptly, the temperature falls to normal within a few hours, and the other symptoms rapidly subside.

The term *variola cornea* (or *horn-pox*) has been applied to those cases in which the eruption does not pass beyond the papular stage, and *variola verrucosa* (or *wart-pox*) to those in which the papules last longer, and become, some of them at any rate, vesicles which quickly dry up.

The various forms of modified or abortive small-pox occur in persons who are partially protected by (i) a previous naturally acquired attack of small-pox, whether confluent or discrete, most commonly the latter; (ii) an attack of small-pox given by inoculation; here again the attack of small-pox may have been severe or mild; (iii) vaccination. It must not be forgotten, however, that modified variola may occur, though rarely, in those who have not been protected in any of these ways. In an unprotected community there will be found a certain number of persons who are altogether insusceptible to small-pox, and others who are but partially susceptible, and in whom the disease shows itself in a modified form.

It is important to remember that modified small-pox is nevertheless small-pox, and that such a case may give rise in other persons to the ordinary forms, discrete, confluent, or even hæmorrhagic.

Complications.—In severe cases complications are common. The most important are *bronchitis*, and *lobular*

pneumonia. These are both frequent and dangerous, and are met with especially during the period of secondary fever. Less frequent are *lobar pneumonia*, *pleurisy*, *empyema*, *œdema of the larynx*, *laryngitis*, *ulceration of the larynx*, *perichondritis* and *necrosis of the cartilages*. *Diphtheria* is rare. *Adenitis* is common.

Though pocks do not usually form upon the conjunctiva, *conjunctivitis* is of frequent occurrence; and it may lead to a corneal ulcer and corneal opacities. A corneal ulcer may perforate and give rise to hypopyon or to panophthalmitis. Serious eye complications, which were formerly very frequent are, nowadays, owing to appropriate treatment, of rare occurrence.

The skin and subcutaneous tissues are liable to various inflammatory conditions. Sometimes the inflammation is erysipelatous, and sloughing may result. *Boils* and *abscesses* are common. *Bedsores* readily form. *Otitis media*, with its results, is not uncommon. During the acute stage of the disease, especially in the confluent variety, extreme *swelling of the tongue* may occur. *Cardiac* complications are uncommon. *Albuminuria* may be present during the febrile stage, but *nephritis* does not often result. Some writers have described certain forms of *paralysis* as sequels of small-pox; the cause is most probably a degeneration of the peripheral nerves. *Septicæmia* is a complication very likely to arise during the period of secondary fever. *Pyæmia*, which was formerly of frequent occurrence, is now rare.

HÆMORRHAGIC SMALL-POX. (*Malignant small-pox*, *purpura variolosa*, *variola nigra*.) There are three conditions under which hæmorrhage may occur into the skin during the course of small-pox; and as these are of quite different signification they must be carefully distinguished from one another.

Firstly, there is the prodromal "triangular rash" which is generally minutely petechial, and is only of importance from the point of view of diagnosis.

Secondly, during the late pustular stage of the variola eruption hæmorrhages are not infrequent into the pustules, and are not of serious import; for, when occurring as late as the ninth day of the disease, even when extensive, they do not in any way affect the prognosis.

Lastly, there is the condition known as hæmorrhagic small-pox, which is one of the most serious conditions that can arise during the course of the disease. It generally comes on during the eruptive, but may appear during the prodromal stage. The most usual time for its occurrence is about the third day of the rash (fifth of the disease). But at whatever stage it appears its manifestations are similar; with the exception that during the eruptive stage hæmorrhages occur in some of the vesicles or into their bases, and, indeed, may be almost confined to them. The hæmorrhagic symptoms are most striking and severe when they occur during the prodromal stage. In this event the ordinary prodromal symptoms are usually exaggerated; one of the initial rashes already described is usually present, and the course of the disease is more rapid than when the hæmorrhage first appears at a later date. It may be so rapid as to terminate fatally before the papules of the ordinary eruption appear.

The symptoms characteristic of hæmorrhagic small-pox are as follows. A purpuric eruption breaks out, consisting of purplish-red spots of various sizes, and of hæmorrhages more or less extensive into the subcutaneous tissue. The skin around the eyes often becomes black, and there may be subconjunctival ecchymosis, the blood being of a brighter hue in the subconjunctival than in the subcutaneous

tissue. Hæmorrhages frequently occur from one or all of the mucous membranes, causing epistaxis, hæmoptysis, hæmatemesis, metrostaxis, hæmaturia, and melæna. Blood may even flow from the eyes or ears. When bleeding occurs from the mouth and fauces putrefactive changes occur, and the breath becomes horribly offensive. The patient rapidly becomes extremely collapsed, and the pulse hardly perceptible. The temperature is not very high, and is rarely above 104° , except sometimes just before death. Generally the patient is quite conscious, and able to converse even up to within a short time before the fatal termination, which usually takes place in three days after the onset of the hæmorrhagic symptoms. Recovery practically never occurs.

The symptoms are the same whether the purpura develops during the eruptive stage or earlier.

Protection.—An attack of small-pox confers a very marked immunity against a subsequent attack; nevertheless second attacks sometimes, but rarely, occur.

The protection afforded by vaccination, and the manner in which it modifies the disease, is discussed elsewhere.

Length of Infectivity.—The patient may be considered free from infection when the scabs have fallen off. The region on which the scabs separate last are the soles and palms, so that particular attention should be paid to these parts.

Morbid Anatomy.—The pocks on the skin remain visible after death, and can also be seen on the various mucous membranes. They are found in the nose, mouth, pharynx, œsophagus, larynx, trachea, and sometimes in the bronchi, and also on the vulva and vagina. The stomach and intestines are free, except, perhaps, the lower part of the rectum; and no pustules are found in the

urethra and bladder. Where the pocks are confluent on the mucous membrane the appearance produced is like that of a layer of diphtheritic membrane. Pocks do not occur on the serous membranes.

The smaller bronchi are filled with muco-pus, and there is often well-marked broncho-pneumonia.

Fatty changes are often found in the liver, kidneys, heart, and spleen. The latter may be soft and diffuent, or may appear quite normal.

In hæmorrhagic small-pox, in addition to those in the skin, hæmorrhages may be found in all the mucous membranes; ecchymoses are seen on the serous membranes; and there may be extensive hæmorrhages under the capsules of the kidneys, in the mediastinum, in the loose connective tissue of the pelvis and abdomen, and elsewhere.

Sections through the pocks at various stages reveal the following changes. In the papular stage the stratum Malpighii is thickened by a multiplication and swelling of the epithelial cells; the blood vessels of the corium and papillæ are dilated, and there is an exudation containing a few leucocytes in the tissues of the papillæ, which are enlarged. In the vesicular stage the middle layer of cells of the rete Malpighii become separated from one another by spaces containing clear exudation, which by compression converts the cells into thin partitions giving a loculated character to the vesicle. Many of the cells also undergo compression by the accumulation of fluid in their interior. The exudation is at first confined to the margin of the pock, and consequently the latter is umbilicated. At an early stage the exudation is poor in leucocytes, but at a later stage the leucocytes increase in number, and the vesicle becomes converted into a pustule.

In the pustular stage, not only the rete Malpighii, but also the papillæ and corium are infiltrated with leucocytes. When the pustule dries up and is converted into a crust, a new layer of stratum lucidum is formed underneath the pustule from the deeper layers of the rete Malpighii, and healing occurs without scarring. But should the inflammatory changes have destroyed the whole of the rete Malpighii, the new epithelium is supplied from the epithelium at the margin of the pustule, and scarring of greater or less severity results.

In the proliferating epithelial cells appearances can be noted which have been attributed by some to the presence of micro-organisms belonging to the class of protozoa. Other observers consider that these appearances are due to degeneration and dropsy of the cells. In the pustular stage various bacteria can be found in the contents of the pocks. Many of these are harmless saprophytes, but the pyogenic staphylococci are constantly present. The streptococcus pyogenes has also been found in a certain proportion of cases. In the early vesicular stage bacteria appear to be absent.

Pathology.—The nature of the contagion of variola has not yet been determined, although there can be no doubt that it is a living micro-organism. The appearances seen in the epithelial cells of the pocks have been considered to be caused by a cell infection with protozoa which are the cause of the disease. Whether this view is correct future experiments can alone decide. At present the evidence is based entirely upon the interpretation given to the microscopical appearances, and upon certain inoculation experiments upon the corneæ of animals.

The contagion certainly resides in the pocks ; for inoculation with the contents will produce variola in the human

being and in monkeys. The pyogenic cocci found in the pustular stage are due to a secondary or mixed infection, and are probably responsible for the fever of suppuration.

Hæmorrhagic small-pox is probably due to extreme virulence of the true small-pox micro-organism, and is in this respect comparable to malignant scarlet fever. A case of severe confluent small-pox is comparable to one of scarlatina anginosa; for in both cases the severity of the symptoms are, to a great extent, due to the intensity of the secondary invasion with pyogenic bacteria.

Diagnosis. — Small-pox may be mistaken for other diseases either in the prodromal, early eruptive, or late eruptive stage.

1. In the prodromal stage it may be mistaken for lumbago, for influenza, or some other acute febrile disease; and if a prodromal rash is present it may be mistaken for scarlet fever, morbilli, rubeola, typhus, or some non-infective erythema. The diagnosis at an early stage is not by any means easy, and is often impossible. In doubtful cases the patient should be isolated, and a definite opinion deferred until the characteristic eruption has appeared. During an epidemic, severe constitutional symptoms, with pyrexia, vomiting, and lumbar pain, should always arouse suspicion.

Lumbago has been mistaken for small-pox, but the absence of pyrexia and other constitutional symptoms render the distinction easy.

Influenza sometimes closely resembles the early stage of variola. In both there may be severe constitutional symptoms, lumbar pain, and pyrexia. The history of exposure to contagion should be inquired into; and it is often necessary to wait for the eruptive stage before pronouncing an opinion.

When prodromal rashes are present mistakes are frequently made. The "triangular rash" is characteristic; and when it is attended with pyrexia and the constitutional symptoms already described, a positive diagnosis can at once be made. But the eruption may be uniform, and have all the appearances of a scarlatinal eruption; or it may be maculated, and liable to be mistaken for morbilli, rubeola, or typhus fever.

The condition of the throat in *scarlet fever* will serve to distinguish this disease from variola. There may be some reddening of the fauces in variola, but there is not the swelling and exudation met with in scarlet fever. An early appearance of the variola papules upon the palate and pharynx can be readily distinguished from the angina of scarlet fever. In addition to these points of distinction the prodromal scarlatiniform eruption of variola especially affects the extensor surfaces of the limbs, and on the feet often picks out the line of the extensor tendon of the great toe. Nevertheless it must be admitted that the characters of the eruption may be exactly like those of the eruption of scarlet fever.

The maculated prodromal eruption does not usually affect the face, and thus differs from the eruption of morbilli and rubeola. In variola catarrhal symptoms are not usually present, while in *morbilli* they are a marked feature of the disease. In *rubeola* the constitutional symptoms are slight. *Typhus fever* can be distinguished by the character of the eruption, the absence of lumbar pain, and the history of the case.

Various *erythemata* and *drug rashes* can be distinguished by the absence of the constitutional symptoms met with in variola.

The purpuric eruption of hæmorrhagic small-pox, when

it appears at an early stage of the disease, is often difficult to diagnose from *purpura*. The rash itself is similar, but severe constitutional symptoms, pyrexia, and hæmorrhages from the mucous membranes rapidly arise in hæmorrhagic small-pox, while in *purpura* they are either absent or occur some time after the first appearance of the purpuric eruption. In hæmorrhagic small-pox the characteristic "triangular rash" or a scarlatiniform prodromal eruption may be present, and the case rapidly terminates fatally. Even in the most rapid cases the true variolous eruption generally appears before death.

2. During the early eruptive stage small-pox may be mistaken for morbilli or rubeola.

The eruption of *morbilli* appears commonly on the fourth or fifth day, catarrhal symptoms are present, and the temperature continues to rise after the appearance of the rash. In *variola* the eruption appears on the second or third day; and with its appearance the temperature falls, and the constitutional symptoms subside, except in the case of confluent small-pox. Catarrhal symptoms are not, as a rule, present in small-pox. In *rubeola* the constitutional symptoms are slight, the rash appears early, and there is frequently no prodromal period at all. Neither in morbilli nor rubeola does the rash give the same solid sensation to the touch as the small-pox eruption. It is not quite true to say that the papules of morbilli altogether disappear to the touch when the skin upon which they are situated is stretched (the so-called "Grisolle sign"). One of the authors has frequently observed that this sign is fallacious. It must be understood that difficulties in diagnosing *variola* from morbilli or rubeola only arise when the eruption first makes its appearance; for within twelve or twenty-four

hours the small-pox eruption presents characters which are quite easily distinguishable from that of morbilli or rubeola.

3. In the late eruptive stage variola may be mistaken for varicella, acne vulgaris, acne varioliformis, syphilides, iodide or bromide rashes, and glanders. In *acne varioliformis* rather large pustules are seen on the forehead, temples, cheeks, and occasionally on the scalp. Pitted scars result; but there is no preceding vesicular stage. Pyrexia and severe constitutional symptoms are absent both in this form of acne and in acne vulgaris. From *syphilides*, whether papular, vesicular, or pustular, a diagnosis can be made by a careful attention to the history of the case, and the manner in which the eruption has come out. It must be admitted that in some rare forms of pustular syphilides an error in diagnosis can easily be made. In *glanders* a pustular eruption resembling that of small-pox may occur, but is usually accompanied by subcutaneous abscesses; in doubtful cases a bacteriological examination will at once clear up the diagnosis. *Iodide* and *bromide rashes* can easily be distinguished from variola by the history of the case.

The diagnosis between variola and *varicella* is discussed in the chapter dealing with the latter disease.

In doubtful cases of small-pox it is well to vaccinate the patient, for should the vaccination prove successful, then small-pox can be excluded. Further information upon this point will be given in the chapter on Vaccinia.

Prognosis.—The *age* of the patient is of importance in forming a prognosis. The fatality is very high amongst children under five years of age (upwards of 50 per cent.). The minimum fatality is found in children between ten and fifteen; after the latter age the fatality rises again, and continues to increase with advancing age.

The *vaccination* of infants has altered the age-incidence of small-pox, and has lowered the general mortality from that disease in directions which will be pointed out in the chapter on Vaccinia.

With respect to the influence of previous vaccination in individual cases of small-pox, there are two points especially to be borne in mind—the length of time that has elapsed since the vaccination was performed, and the successfulness of the operation, as shown by the number and size of the scars. Vaccination confers an immunity against small-pox, which, however, lessens as time goes on. But even when, through lapse of time, the complete protection against an attack is lost, the power of modifying an attack remains, and remains for a considerable period. The length of time during which the complete protection and the power of modification lasts varies with the nature of the individual and the successfulness of the vaccination. If a vaccinated person is attacked with small-pox, the chances are much in favour of the disease assuming a modified form; and the shorter the time since, and the more efficacious the vaccination, the more likely is the attack to be mild. A reference to the paragraph on modified small-pox will inform the reader to what extent modification occurs. It will suffice to add here that modification may occur even when a copious eruption of papules has appeared, and that the modifying action may be seen even in cases of the confluent form. The case-mortality of confluent variola is very much less in the vaccinated than in the unvaccinated.

Apart from the age of the patient and the influence of vaccination, the prognosis chiefly depends upon the form of the disease. The most fatal variety is the hæmorrhagic, from which recovery very rarely ensues,

death usually occurring in four or five days. The next in order of fatality is the confluent form; roughly speaking, about 50 per cent. of these die. The fatality of semi-confluent cases (according to Marson) is 8 per cent., and of the discrete form 4 per cent. These figures refer to unvaccinated patients. In modified small-pox recovery almost always ensues. It is thus possible to give a fairly accurate prognosis according to the nature of the eruption. Except in the hæmorrhagic form, death does not take place usually before the eleventh day, and the twelfth, thirteenth, and the fourteenth days are those upon which the fatal termination is most often seen.

Patients addicted to alcohol bear an attack of small-pox badly. Insomnia and severe delirium are bad symptoms. Of complications, those affecting the air passages, especially the larynx, are the most dangerous. It is stated that an attack is very likely to end fatally when occurring during pregnancy, abortion usually taking place before the death of the patient; but this is not in accordance with the experience of the Hospital Ships of the Metropolitan Asylums Board.

The question arises as to whether the variety which the disease will assume can be predicted from the initial symptoms. It can be stated that a mild initial stage is almost always followed by a mild attack, but that a severe initial stage may be followed by a severe or by a mild attack.

Treatment.—The patient should be placed in a warm but well-ventilated ward.

During the *initial stage* he will often complain of feeling chilly, and will be benefited by the use of hot-water bottles. The lumbar pain is relieved by poultices or dry cupping. A high temperature, with restlessness or

delirium, is best treated by tepid sponging or by a wet pack. The diet should be light, consisting of beef-tea, milk, etc. When thirst is complained of, lemonade made with fresh lemons or imperial drink may be given with advantage.

During the whole of the *eruptive stage* baths should be given night and morning in all but the most severe cases, the temperature of the bath being 98° Fahr. A solution of permanganate of potash (four grains to the ounce) may be added to the bath.

In severe discrete and in confluent cases the continuous warm bath, as recommended by Hebra, is of great value in allaying the terrible irritation of the skin, and in giving the patient sleep. If suitable arrangements are made for the patient to recline with his head out of the water, and for the emptying and filling of the bath, he may be left in it for days together. When the bath is impracticable, sponging with warm water can be advantageously used; and a cradle to keep the bedclothes off the skin is sometimes of benefit.

The pocks should be treated on antiseptic principles. Either vaseline and iodoform, or carbolised oil (one in twenty), or oxide of zinc ointment, or linimentum calcis, should be applied. A mask of lint, soaked either in boracic acid lotion or in glycerine and water, should be kept on the face. Dr. Birdwood recommends a paste made of Pulv. Cretæ Aromat. (fifteen grains to one ounce of water). The pain and swelling of the hands and face are relieved by the application of iced compresses. Extreme itching during the stage of healing of the pocks is best allayed by sponging the skin with a lotion consisting of one part of dilute acetic acid to three parts of water. The hair should be cut quite short.

Difficulty in swallowing is common in severe cases,

and liquids may easily get into the larynx. Under these circumstances nutrient enemata should be administered until the difficulty is over.

Sore throat is relieved by the sucking of ice or the application of glycerine and borax. For sleeplessness and delirium a warm bath, sponging with warm water, or the wet pack, are the best remedies, and in some cases narcotics are advisable. A delirious patient requires watching by a strong attendant, and a sheet loosely passing over the chest may be fastened to the sides of the bed to prevent the patient from getting out.

The diet should consist of liquid nourishment, milk, beef-tea, soup, cocoa, meat juice, lemonade, etc.

When the period of secondary fever has passed the patient may be put upon fish, and in a few days upon a meat diet. Green vegetables and fresh fruit seem to hasten convalescence.

A mixture containing quinine and iron is useful, more especially during convalescence.

Of the various *complications* the following require special mention under the head of treatment :—

Cleanliness is of the greatest importance in preventing *eye complications*. In former days small-pox was one of the most frequent causes of loss of sight, and this was probably due to the lack of proper care. The lids and conjunctivæ should be frequently bathed with lotions of boracic acid and potassium permanganate. For *suppuration* of the *Meibomian glands* or *hair sacs* the following ointment is useful :—

Hydrarg. ox. flav.	gr. xvi.
Acidi boracici	gr. xx.
Cocain hydrochlor.	gr. x.
Vaseline	℥ i.

In *conjunctivitis* the eyes should be thoroughly syringed every few hours night and day with boracic acid lotion, or with corrosive sublimate (1 in 6,000). *Corneal ulcer* is treated by the local application of atropine; if necessary the ulcer may be touched with solid silver nitrate.

Boils and *abscesses* should be opened early, and treated on antiseptic principles. *Bronchitis* is usually of sufficient intensity to require treatment, and the usual remedies for this condition should be used. If symptoms of *laryngeal obstruction* arise tracheotomy may be necessitated.

Hæmorrhagic small-pox is unfortunately almost invariably fatal in spite of treatment; but a mixture containing twenty to twenty-five drops of oil of turpentine, and half a drachm of liquid extract of ergot, may be given every three or four hours; ice should be applied locally to the bleeding parts, and stimulants administered as required.

In the treatment of small-pox, isolation, preferably in a special hospital, should be rigidly enforced. The sooner the patient is removed to a hospital the better, although the infectivity during the initial and the beginning of the eruptive stage is not so pronounced as in the vesicular and pustular stages. All those who are living in the same house as the patient, or who have been brought in contact with him, should be urged to be revaccinated if they have not recently undergone the process. Successful vaccination or revaccination will usually ward off or modify an attack of small-pox in a person who has been exposed to infection, if performed during the first three days from exposure; and if delayed till the fourth, fifth, or sixth day, will usually modify the attack.

The house and all articles that have been in contact with the patient should be thoroughly disinfected. Care

should be taken that those who carry out the process of disinfection are protected by revaccination.

A small-pox hospital should be situated in an uninhabited locality; for it has been distinctly shown that such a hospital forms a focus of disease to the neighbourhood, especially if the latter be crowded and the hospital contains many acute cases.

CHAPTER XI.

VACCINIA.

IMMUNITY to an attack of small-pox can be acquired in three ways: (1) By a previous attack of natural small-pox; (2) By the inoculation of small-pox, or variolation; (3) By vaccination.

The most perfect protection is obtained by a *previous attack* acquired in the natural way; but such protection is not absolute, for second and even third attacks do occur. It would appear that the more severe the primary attack the greater is the protection afforded, and that second attacks are generally modified.

Variolation is now forbidden by law, so that we need say but little about it. Before the introduction of vaccination it was common, and there is no doubt that it conferred a considerable amount of protection. The operation was performed by introducing the contents of a small-pox vesicle from another patient either under the skin or into the nostrils. A local pustule formed, and this was followed on the ninth or tenth day after the operation by constitutional symptoms, and on the eleventh to the thirteenth day by the specific eruption of normal or modified small-pox. The disease thus induced differed from natural small-pox only in being less severe; it was infectious, and gave rise to ordinary small-pox in other

patients, and in a certain proportion of cases it ended fatally.

Vaccination consists in inoculating a person with (1) lymph from the vesicles of cow-pox, a natural disease of the udder of cows; (2) lymph obtained by transmission of cow-pox through either the human subject or the calf; (3) lymph obtained from the vesicles produced by inoculating calves with the contents of a small-pox vesicle from the human subject.

Cow-pox—that is, the disease occurring naturally on the udders of cows—is not so common as it was at the beginning of the century; besides, there are other diseases affecting the udder, not readily distinguishable from true cow-pox. Consequently the first method of vaccination is not at present in vogue.

The lymph now in use was derived originally from cases of cow-pox, and has either been transmitted through the human subject (humanised lymph, arm to arm vaccination), or through calves (bovine or calf lymph). In this country calf lymph is supplied by the Local Government Board, who have established a station in Lamb's Conduit Street, Bloomsbury; and by Dr. Hime, of Bradford. The original source of the lymph in the former instance was a case of natural cow-pox occurring at Lafôret, near Bordeaux; and the supply has been kept up by transmission from calf to calf, without ever having been passed through the human subject. The calves are inoculated on the shaven skin of the abdomen, and the lymph, for the purposes of vaccination, is taken from the vesicles on the fifth day. The lymph is stored in a manner shortly to be described, but does not keep its potency very long, and should therefore be used as soon as possible after being received from the station.

The supply of humanised lymph is kept up at a number of stations in various parts of Great Britain by vaccinating one child directly from another, and when necessary storing the lymph.

Lymph has also been obtained by inoculating calves with the fluid from small-pox vesicles in the human subject; and after one or more passages through the calf it produces the same effects as the lymph obtained in the ways above-mentioned.

The operation of vaccination is performed in various ways. Scratches or punctures are made in the skin with lancets or needles previously charged with lymph, or abrasions or scarifications are first made, and the lymph then rubbed in. A slightly blunt lancet is better than a sharp one for making the scarification. The skin should be carefully cleansed before the operation, and the lancet sterilised by holding it in the flame of a spirit lamp before charging it with the lymph. Only the tops of the papillæ of the skin should be laid bare, and it is best not to cause bleeding, in order that the lymph should not be washed away from the wound by the flow of blood. The spot usually selected for the site of vaccination is the outer side of the left arm just below the insertion of the deltoid muscle. The lymph should be inserted in four spots half an inch from one another, so that four large vesicles or collections of vesicles may be formed. When the lymph has been inserted or rubbed in, the scarification should be allowed to dry before the arm is again put in the sleeve, and should not be washed until the vaccination has taken or failed.

Certain precautions must be observed in performing the operation. As a slight wound is produced in the skin, and as slight wounds, from whatever cause, are occasionally

the starting-point of erysipelatos or other inflammatory conditions, the instruments should be kept scrupulously clean, and should be sterilised before use. The person, whether child or adult, should be in good health at the time of vaccination, and especial attention should be directed to the skin to ascertain that it is not inflamed nor in any way diseased. The only exception to this rule would be instances where patients in a hospital ward have been exposed to the infection of small-pox ; even then there may be some patients whom it will be best not to vaccinate. In cases where the time for vaccination can be selected, the operation should not be performed when there is any risk of the person being exposed to the contagion of erysipelas or other septic diseases. In arm to arm vaccination the child from whom the lymph is taken (the vaccinifer) should be in perfect health, and especial care should be taken that there are no signs of syphilis, tubercle, or skin disease. It is also well to ascertain that there is no hereditary disease in the child's parents. The lymph should be taken from fully developed uninjured vesicles, which have no red areola around them. It should be quite transparent and clear, not purulent, nor, on the other hand, too watery. Care should be taken that no blood gets mixed with the lymph when removed from the vesicles. Only lymph derived from cases of primary vaccination should be employed, never from cases of revaccination.

In cases where stored lymph is used for vaccination the source of the lymph should be known. Lymph is stored by being dried on ivory points or collected in previously sterilised capillary tubes, which are inserted into the vesicle and, when filled by capillary attraction, sealed in a spirit lamp. It can also be stored by mixing with

glycerine in sterilised vials. Stored lymph becomes inert after some time.

The phenomena of vaccination.—The following are the appearances to be observed in a case of successful primary vaccination either with humanised or calf lymph. By the end of the third day (counting the day of inoculation as the first) a red spot appears at the seat of inoculation; this spot quickly becomes a papule, upon which by the fifth or sixth day a vesicle forms with a depressed centre and raised edge. The vesicle increases in size up to the eighth day, and is then distended with quite clear and transparent lymph; and it is on this day that lymph should be removed for storage or for the immediate vaccination of other persons. A red zone now appears round the vesicle, and increases in size for the next two days so as ultimately to extend from one to three inches beyond the vesicle; at the same time the skin and subcutaneous tissue become indurated. In the meanwhile the lymph has become opaque and thick; but on the tenth or eleventh day the inflammation begins to subside and the vesicle begins to dry up and turn of a brownish colour. By the fourteenth or fifteenth day a hard reddish-brown scab has formed, which gradually contracts and separates from the skin, falling off about the twenty-first day. A cicatrix remains which is at first pink, but in course of time becomes white; it is slightly depressed, usually circular in shape, and is dotted with small indentations (foveated). The total area of the cicatrices should not be less than half a square inch.

Constitutional symptoms are generally present from the fourth to the tenth or eleventh day—viz., feverishness, restlessness, and gastro-intestinal disturbances. A papular (lichenous) or vesicular (herpetic) eruption may

appear on the limbs, and to a less extent on the trunk, on the eighth or ninth day, and lasts from four to seven days. In some cases the local inflammation at the seat of vaccination is more severe than has been above described, and there may be much swelling of the upper arm with enlargement of the glands in the axilla.

Care should be taken that the vesicles are not accidentally ruptured, and that the scab is not picked or rubbed off prematurely, otherwise the pocks may take on an unhealthy action, and large ulcers may form. In cases where the local phenomena are severe, salicylic cream may be smeared over the inflamed skin, and the parts wrapped in salicylic wool.

It is very uncommon for an attempt at primary vaccination to fail if good lymph is employed. A failure should arouse suspicion as to the efficacy of the lymph, and a second attempt should be made with a fresh supply. Failure after a second attempt with good lymph is rare.

Revaccination.—By this term is meant vaccination in a person who has been already successfully vaccinated. The greater the length of time since the primary vaccination, the more likely is a revaccination to prove successful. After a primary vaccination a person may be protected against an attack of small-pox at a time when he is still susceptible to revaccination. The local result of revaccination—supposing the lymph to be good—may be (1) similar to that of primary vaccination; (2) modified reaction; (3) negative. These different results depend upon the amount of protection afforded by the primary vaccination. Modification consists either in the pock not going beyond the papular stage, or in an irregular shape of the vesicle, which passes through the various stages more rapidly than usual.

The zone of inflammation around the vesicle and the swelling are prone to be more extensive than in primary vaccination. It is advisable to revaccinate children at the age of ten or twelve years, for by this time the protection afforded by the primary vaccination has considerably diminished.

Those who are much exposed to the contagion of small-pox should be vaccinated at least every ten years. If an attempt at revaccination should fail a second or even third attempt should be made, and unless there is evidence from the successful vaccination of other persons that the lymph is good, a fresh supply must be obtained.

Complications of Vaccinia.—*Erysipelas*, and *cellulitis* are sometimes set up by vaccination, as indeed they may follow upon any abrasion of the skin. They may be due to a dirty lancet, to the use of contaminated lymph, or to infection occurring subsequent to the operation. The frequency of such complications is reduced to a minimum by aseptic precautions in performing the vaccination, by care in the selection of the lymph, by preventing the vesicle from being rubbed, and by scrupulous cleanliness. *Impetigo contagiosa* may also follow vaccination, but can be prevented by not allowing the child to pick the scab.

A number of *rashes* have been attributed to vaccination. In the majority of cases the vaccination has nothing to do with these eruptions ; but there are others which owe their origin to this cause. They are :—

I. *Vaccine Lichen*, a papular eruption, usually coming out on the eighth day, but not infrequently earlier or later. The papules are of a bright red colour, small, and acuminate ; vesicles and pustules may appear. The eruption generally begins on the arms, though the face, trunk, or neck may be the parts first affected. It spreads

by successive spots over the whole of the body, and lasts for a few days, a week, or even longer.

2. *Roseola Vaccinia*, consisting of a uniform or morbilliform erythema, has been described by various authors as occurring from the third to the eighteenth day.

3. *Generalised Vaccinia*.—Rare cases have been recorded in which lesions similar to the primary vaccination have formed in various regions of the body.

Diagnostic value of Vaccination.—Vaccinia may run its course concurrently with small-pox. In the report of the Committee of the Clinical Society on Incubation will be found notes of twenty-two cases in which successful vaccination or revaccination was performed at times varying from thirteen days before the outcome of the eruption of small-pox up to, in one case, the very day of its appearance. In this case successful vaccination was performed a few hours before the outcome of the small-pox eruption; but this appears to be quite an exceptional occurrence.

During the eruptive stage of small-pox, during convalescence, and for a long period afterwards, successful vaccination is impossible.

In a doubtful case vaccination should be performed, for, if successful, the case is almost certainly not variola. But the converse of this does not hold; a failure to produce a successful vaccination is of no value from a diagnostic point of view.

Nature and Pathology of Vaccinia.—There has been much controversy as to the nature of vaccinia. Is cow-pox a distinct disease from small-pox, or is it the same disease altered in character by transmission through the cow? The problem is not an easy one to solve for several reasons. In the first place, it is difficult to inoculate a cow successfully

from a case of small-pox in the human subject. Many experiments have been made, but only few have given positive results. It would appear that two phenomena may arise in those cases in which any effect has been produced. Either papules running a rapid course are formed, or vesicles arise having all the appearance of those of cow-pox. In the latter case similar lesions have been transmitted to other cows by inoculation with the contents of these vesicles, and from these lesions children have been inoculated, with the result that a typical vaccinia has been produced. We consider such evidence as a sufficient proof of the identity of small-pox and cow-pox. On the other hand, there are cases recorded where children have been inoculated with the lymph from the lesions produced by the inoculation of cows with small-pox, with the result that they have contracted small-pox and not vaccinia. But in some, at any rate, of these cases the lesions in the cow were those of papules rapidly disappearing; and it is probable that the children were inoculated with some of the small-pox virus originally introduced, and which had remained unaltered at the seat of inoculation in the cow, as was evidenced by the absence of typical vesicles in this animal. This is indeed what we should expect, for the virus would probably not become fixed in its new character until it had been transmitted through several cows.

The microscopical changes observed in vaccinia are similar to those found in variola in the human subject, and the same appearances supposed to be due to infection of the cells by protozoa have been observed. Various saprophytic bacteria have been found in the lymph, but appear to have no bearing upon the pathology of the affection.

The Value of Vaccination.—It is unnecessary in a work

of this kind to set forth at length all the arguments showing the great value of vaccination as a protection against small-pox. There are two ways in which vaccination protects. The one is by preventing the occurrence of the disease, and the other by mitigating the severity of the attack.

The chief arguments showing the preventive power of vaccination are the following :—

(i) When variolation was in vogue a large number of vaccinated and unvaccinated persons were subjected to this test, and it was found that, while the unvaccinated persons almost invariably took small-pox when inoculated, those who were vaccinated almost invariably escaped.

(ii) Since the introduction of vaccination the disease, as shown by the mortality statistics, has enormously declined.

(iii) In the pre-vaccination days children under five years of age were mostly attacked, and amongst them the mortality was most severe ; nowadays, in countries where vaccination is practised, the children mostly escape, and it is the adults who suffer. This alteration in age incidence is doubtless due to the protection afforded by vaccination, which declines as time elapses.

(iv) In outbreaks of small-pox the attack rate is much higher amongst the unvaccinated than amongst the vaccinated.

A striking instance of the value of revaccination was brought out by the Vaccination Committee of the Epidemiological Society with regard to 734 attendants in the Metropolitan Asylums Board Hospitals. Of these, 79 had had small-pox and 645 had been successfully revaccinated before entering upon duty, and none of them were attacked ; the remaining 10 had not been revaccinated,

and all contracted the disease. Again, in Prussia since revaccination was made compulsory small-pox has almost entirely disappeared ; and many more instances might be quoted.

The influence of vaccination in modifying the severity of an attack of small-pox is shown by a number of mortality tables comparing the mortality among the vaccinated and unvaccinated. In Marson's figures, for instance, in 1852-67 the mortality among the unvaccinated was 34·9 per cent. ; among those stated to have been vaccinated, but with no cicatrix, 39·4 per cent. ; among those with one cicatrix, 13·8 per cent. ; among those with two cicatrices, 7·7 per cent. ; among those with three cicatrices, 3·0 per cent. ; and among those with four or more cicatrices, 0·9 per cent. These figures, which have been completely corroborated by similar evidence brought forward by Dr. Gayton, show that the more efficiently the vaccination has been performed the greater is the protection afforded.

Objections to Vaccination.—The principal objections against the practice of vaccination are the following :—

(i) That vaccination does not protect against small-pox. We have already stated all that is necessary in regard to this objection.

(ii) The risks incidental to vaccination, especially those of incurring syphilis, erysipelas, leprosy, and a variety of other affections. Now there is no doubt that erysipelas and syphilis can arise from vaccination. With regard to syphilis the risk is infinitesimal, only a few cases (four or five) having occurred in England. By using calf lymph this slight risk is entirely avoided. The risk of erysipelas is also very slight, and is very much less than that run by being unprotected from the danger of contracting small-pox. As to the other diseases, such as

tuberculosis, said to be caused by vaccination, there is no evidence that they are caused in this way.

(iii) Another objection to compulsory vaccination is of a political nature, and one with which we are not now concerned. It should, however, be pointed out that the only other method of preventing the spread of small-pox is by compulsory isolation and quarantine, which would appear to be more open to this kind of objection than is vaccination.

CHAPTER XII.

VARICELLA. CHICKEN-POX.

VARICELLA is a febrile disorder, rarely severe, of which the characteristic symptom is an eruption of vesicles.

Etiology.—The disease is met with chiefly in children, especially in those under five years of age, but it is occasionally seen in adults. It occurs in localised outbreaks, but sporadic cases are often observed ; unlike variola, it does not assume the form of large epidemics. It is slightly more prevalent during the months of October and November than at any other season of the year. It is very infectious, and is spread chiefly by the direct exposure of the healthy to the sick ; but the infection may certainly be conveyed by fomites, and by third persons.

Until quite recently there have been those who believed that varicella is a mild form of small-pox. The reasons against this view are numerous. A person may have each of these diseases within a very short time, the one not protecting against the other.* Vaccination has a protective influence against variola, but not against varicella.

* For an instance see p. 35.

One case of varicella gives rise to another case of varicella, and not to any form of small-pox ; whereas even a modified case of small-pox may give rise to severe unmitigated cases of the same disease. If varicella were mild or modified small-pox, it should be found to give rise to undoubted cases—not necessarily mild or modified—of this affection. Varicella was described and differentiated from small-pox in England long before vaccination was introduced into practice—that is, before cases of modified small-pox became very common. The epidemic and clinical histories of the two diseases are quite different. Small-pox is easily inoculable in persons who are unprotected, but varicella with difficulty, if at all. There can therefore be no doubt whatever of the duality of the two diseases.

The **incubation period** is variable, being from twelve to nineteen days.

Clinical History.—The *period of invasion* is very often wanting, the eruption being the first symptom observed ; but occasionally for twelve to twenty-four hours before the appearance of the rash the patient is fretful and out of sorts, and the temperature may be raised two or three degrees. Rarely there is a *prodromal rash* during this period. It consists of a uniform red blush upon the chest, sometimes extending over the whole trunk.

The *eruption*, which makes its appearance usually on the first, never later than the second day, begins as rose spots which are but slightly raised, and fade on pressure. They are not unlike the spots seen in enteric fever, but are of a deeper shade. Very quickly they are converted into vesicles. It is probable that some vesicles form upon healthy skin without preceding papules. At any rate it is not uncommon to observe vesicles where a few hours before no papules were present. The vesicles are circular

or oval in shape, and an eighth to a quarter of an inch in

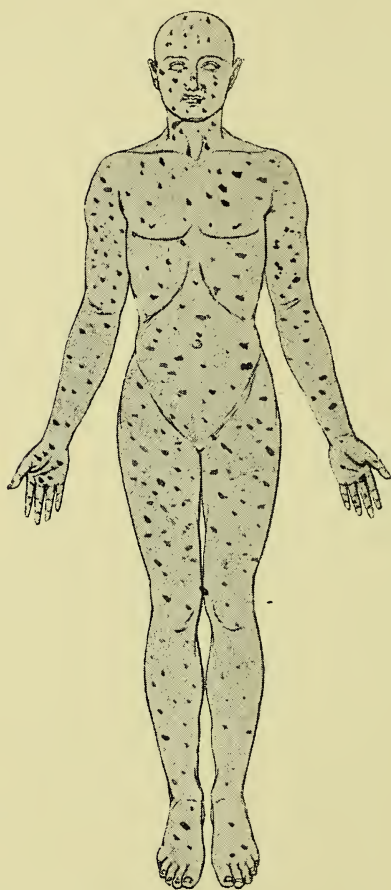


DIAGRAM X.—VARICELLA.

A papular and vesicular and occasionally pustular eruption. The whole of the skin may be involved. When the pocks are numerous, they are to be seen more upon the trunk than the extremities and head, and more upon the proximal than the distal portions of the limbs.

diameter. Some are surrounded by a faint red zone, some surmount a papule, and others are wanting in these

features, looking like drops of water upon the skin. Some are slightly depressed in the centre (umbilicated), especially

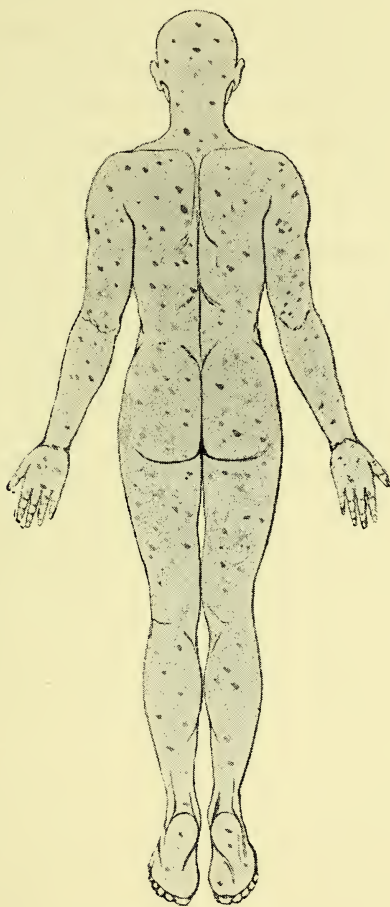


DIAGRAM XI.—VARICELLA.

those that have formed round a hair-follicle, or the opening of a sweat-gland. The vesicles collapse entirely when pricked. The fluid which they contain, at first clear,

becomes after a few hours slightly turbid. At the end of twelve or twenty-four hours from their appearance the vesicles begin to dry up, and thin brownish-yellow scabs are formed. In five to seven days the scabs fall off, leaving pink marks, which gradually disappear. It is not uncommon for a few of the vesicles to become pustular. Pustulation takes place quickly, within two or three days or even one day from the appearance of the vesicle. There can also usually be found a few vesicles, which are very minute, and situated on the summit of distinct papules.

The eruption comes out in crops for three to five days, occasionally even for a week, so that all stages of the eruption can be observed at the same time. The spots come out first upon some part of the trunk, often on the buttocks; then they appear on other parts of the trunk, the scalp and face, and the extremities. There is no definite sequence of invasion of the various parts. Occasionally the spots are seen first upon the face. They are usually not so abundant upon the face and extremities as upon the trunk, and they occur in larger numbers upon the proximal than upon the distal portions of the extremities. They may be found upon the palms and soles, and in these places the spots often do not develop beyond the papular stage. On the palms, soles, and scalp both papules and vesicles may feel "shotty."

Some of the spots never reach the vesicular stage; indeed, some of them may be only slightly or not at all papular. The eruption gives rise to much itching, so that the pocks are often scratched, and slight bleeding and ulceration result. In those cases in which pustulation has taken place in any of the pocks scars may be left.

The spots are often found upon the palate, less frequently upon the tongue, mucous surfaces of prepuce and labia

majora, and the palpebral conjunctiva. When they occur upon the prepuce and labia majora swelling of these structures may take place and lead to painful micturition. A pock upon a mucous surface has at first the appearance of a small whitish patch surrounded by a red zone; later a superficial ulcer replaces the patch.

Course of the Disease.—While the eruption is coming out the child is peevish and irritable, and does not sleep or take its food well. In very mild cases the temperature is not raised; but when there is much eruption it may rise two or three degrees above the normal, remain up for a short time, then fall to rise again later. In exceptional cases the temperature may be continuously up to 100° or 102° Fahr., or even higher, for three or four consecutive days, after which it quickly falls. Even in these cases the constitutional symptoms are by no means severe. Occasionally, however, when the eruption is very copious, in addition to pyrexia there are a frequent and small pulse and quickened respirations, and the patient may be delirious. In children who are weakly or whose health is undermined by some previous illness, death may result; though, apart from the gangrenous form of the disease to be described later, this termination is very rare.

The **complications** of varicella are very few. On the scalp a troublesome *impetiginous inflammation* may be the result of the eruption, especially if treatment has been neglected; and local *eczema* may be set up in other parts by scratching and want of cleanliness.

According to some authors *acute nephritis* may arise during convalescence; but this sequel is rare.

Varicella Gangrenosa.—This is a grave condition, happily uncommon, which is by some accounted a variety

of varicella, but is perhaps better looked upon as a complication. The attack having commenced in the ordinary way, the eruption declares itself as usual; but some of the pocks become angry looking, a dark red zone of inflamed skin appears around them, and their

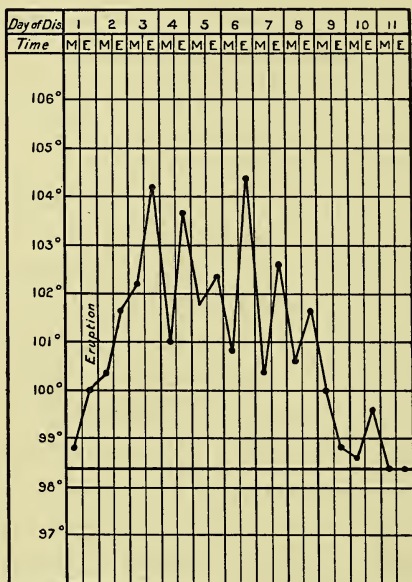


CHART Q.

Boy, aged 3. Severe uncomplicated case of varicella. Rash appeared about noon on first day. The pocks were very abundant by the fourth day, and fresh ones continued to come out till the eighth.

contents become pustular. The zone and corresponding pock increase in size, so that the diameter of the pock may be three-quarters of an inch. In the course of the next two or three days the pock is replaced by a thick reddish-brown or black scab. If the scab be detached its under surface is found to be composed of

sloughing tissue. When the slough separates a circular ulcer with clean-cut edges, looking as if it had been punched out of the skin, results. The depth of the ulcer varies; on the scalp it may go down to the bone. The base of the ulcer is unhealthy, and has a tendency to

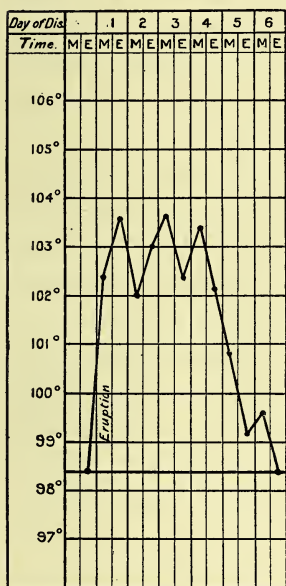


CHART R.

Boy, aged 3. Sharp attack of varicella; rash early on first day.

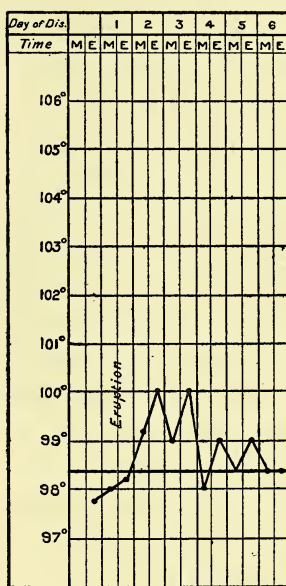


CHART S.

Boy, aged 4. Varicella, with profuse eruption, yet slight pyrexia. On the face the rash was very like variola.

bleed. Meanwhile, the child becomes seriously ill and wastes, and the temperature is raised several degrees. Such cases are often fatal; but if only a few of the pocks have taken on the gangrenous process recovery takes place. Together with the sloughing pocks normal vesicles are to be found. If any of the pocks on the palate

are thus affected an appearance very like diphtheria is observed, and there may be much œdema.

There is another rare variety of chicken-pox called *Varicella Bullosa*. In this form, in addition to the ordinary vesicles, bullæ are to be seen, which may have a diameter of nearly two inches. The bullæ have very thin walls, and contain a fluid which is at first clear, but rapidly becomes turbid. They soon burst, and leave excoriated patches. The constitutional symptoms are more severe than in the common form of varicella.

It has previously been stated that occasionally some of the papules of varicella do not become vesicular, and that in others the vesicle is very imperfectly developed. In certain cases the whole of the eruption may thus abort, and the disease may then be said to be *modified*.

Relapses and second attacks, if they occur, are very rare.

Length of Infectivity.—A patient is infectious from the commencement of the disease until all the scabs have fallen off.

Of the **morbid anatomy** there is little to be said. Patients rarely die of chicken-pox; when they do, it is of the gangrenous form. In some cases of this kind tuberculous lesions have been found post-mortem. Possibly in these instances the presence of tubercle has determined or favoured the occurrence of gangrene. When death has occurred from some other disease during an attack of varicella, there has been no lesion observed post-mortem other than the papules, vesicles, or pustules observed during life.

Pathology.—Very little is known about the pathology of varicella. The contagium is certainly a living micro-organism, and appearances in the epithelial cells of the pocks have been attributed to an infection with protozoa.

Diagnosis.—The importance of varicella with respect to diagnosis lies in the fact that it is so often mistaken for small-pox, or the latter for it.

From most cases of discrete, and still more of confluent, small-pox the differentiation is easy. The well-marked prodromal period of nearly three days, the length of time the eruption takes to mature, the order in which the different portions of the skin are invaded, and the “shotty” character of the variolous rash in its papular stage, are features absolutely different from those of varicella. But the diagnosis between varicella and certain cases of modified small-pox is often extremely difficult; in fact, in some cases it may be impossible; and it is well to admit this, because a dogmatic expression of opinion may lead to serious consequences. Thus, if in a community not at all or indifferently protected by vaccination, a case of modified small-pox were pronounced to be varicella, and were not to be isolated by reason of its supposed triviality, such a decision would almost certainly result in the occurrence of other cases of small-pox, some of which might prove fatal. Any doubtful case, therefore, should be treated as one of small-pox, although it should not be removed to a small-pox ward.

The points of difference between the two diseases are as follows:—In varicella there is often no prodromal period, or if there be, it is not longer than twenty-four hours; the prodromal period in modified small-pox is usually present, and of two or three days’ duration. In varicella with the appearance of the rash the patient usually feels ill, even though slightly, and continues to be so while the rash is coming out; in variola (mild or modified) the feeling of illness disappears with the outcome of the rash. In varicella the temperature may be elevated

while the eruption is making its appearance ; in variola it is raised during the prodromal period, but falls with the onset of the eruptive stage. In varicella the rash usually appears first on the trunk, in variola on the face. In varicella the eruption comes out in crops for three to five days, or more, in various parts of the skin ; in variola it takes not more than three days to come out, and invades the skin in a definite sequence (face and scalp, wrists, arms and trunk, legs). The distribution of the rash in variola is definite, the face, hands and forearms, back of trunk, feet and legs, being more affected than other parts, though for modified small-pox this statement does not hold good. In varicella, when there is much eruption, the trunk is more affected than the extremities, and the proximal parts of the extremities more than the distal. In variola, papules and vesicles, or vesicles and pustules may be present at the same time in different regions of the body, but not side by side in the same region, as in varicella. In varicella the spots become vesicular within a few hours, and pustular within two or three days, when pustulation results ; in small-pox, even when modified, vesiculation begins not earlier than twenty-four hours from the commencement of the eruption. The papules of varicella are not so firm, and do not implicate the skin so deeply as those of variola. In both diseases minute vesicles surmounting papules may be seen. Lastly, in this country at the present time variola is most often met with in persons above ten years of age, whereas varicella is chiefly a disease of children under ten. The scars of small-pox are foveated or pitted, while those of varicella are irregular, smooth, and have a punched-out appearance.

Varicella bullosa may be mistaken for *pemphigus*, but

can be distinguished by the presence of ordinary varicella vesicles in addition to the bullæ, by the clinical course of the case, and by a history of infection.

The **prognosis** is almost always good. It is only in very exceptional cases and in the gangrenous form that an unfavourable termination is to be feared. This variety occurs in badly fed, ill-cared-for, and unhealthy children.

Varicella is said to be sometimes followed by tuberculosis.

Treatment.—There is little to be said under this heading. The child should be prevented from scratching or picking the pocks, by putting fingerless gloves on the hands. If there be an abundant eruption on the scalp the hair should be cut short.

In the gangrenous form quinine should be administered, together with some stimulant. The thick scabs and sloughs should be removed by poulticing, and antiseptic dressings applied to the ulcers.

A patient recovering from varicella should be isolated till all the scabs have fallen off.

CHAPTER XIII.

WHOOPIING-COUGH. PERTUSSIS.

WHOOPIING-COUGH is a disease of about two months' duration, of which the most characteristic features are catarrh of the bronchial tubes, and a frequent paroxysmal cough.

Etiology.—The disease, though occurring in all parts of the world, is found to prevail mostly in the temperate regions. In England and Wales it is, among the zymotic diseases, next to diarrhœa, the cause of the largest number of deaths in children.

It is met with both sporadically and in the form of localised outbreaks.

It has been stated by some authors that whooping-cough has some close relationship with measles, an attack of the one disease being often soon followed by an attack of the other. But any such association is probably only accidental, since both are very common disorders.

Season.—In London the smallest number of deaths from whooping-cough occurs in September. The number begins to rise steadily in November, attaining a maximum in March. In April the numbers begin to decrease, and the minimum is again reached in September.

Dissemination.—Pertussis is almost entirely spread

directly from patient to patient, but there is reason to believe that the virus may be harboured in fomites.

Age.—Though no age is absolutely exempt from whooping-cough, yet it is essentially a children's disease, most of the cases being met with in children under seven years of age.

The **incubation period** is variable, five to fourteen days.

Clinical History.—The course of the disease may be divided into two stages, the "catarrhal" and the "paroxysmal."

The *catarrhal stage*.—The attack begins insidiously. The child is out of sorts, has slight pyrexia, and a dry, harsh cough. Examination of the chest reveals the presence of bronchitis. This stage lasts about ten days (but it may be of longer or shorter duration), and then the characteristic paroxysmal cough appears.

The *paroxysmal stage*.—The paroxysm of coughing, which is characteristic of the disease, is sometimes preceded by a short period during which the child is restless and anxious. The paroxysm consists of a series of short suffocative coughs, interrupted at intervals by a crowing inspiration or "whoop." As the disease progresses the paroxysms become longer and more violent, and frequently terminate by the vomiting of food and blood-stained mucus. During a severe paroxysm the child sits upright, the veins of the face and neck become distended, the face becomes swollen, and tears roll from the eyes. As many as six whoops may occur in one paroxysm. After the attack the child lies back quite exhausted. Occasionally a paroxysm begins with a deep inspiration instead of the usual short expiratory coughs. The number of paroxysms varies from ten to thirty in the twenty-four hours. From some of them the characteristic whoop may be absent,

and this is especially the case at the beginning or towards the end of the paroxysmal stage.

The attacks usually come on spontaneously, but they may be set up by crying or screaming, or even by examining the pharynx. Between the attacks in an uncomplicated case the child seems pretty well, and plays about as usual. Auscultation reveals nothing except occasional râles.

After a time the frequent recurrence of the paroxysms causes the face to be swollen and of a slightly dusky hue; the eyes are watery; minute hæmorrhages and congested patches are found in the skin of the face, and occasionally subconjunctival ecchymosis.

Not infrequently when the characteristic paroxysm with its whoop has long disappeared, a fresh bronchitis, accidentally arising, will be accompanied by a similar paroxysmal cough, and even by the whoop.

The disease usually lasts six or eight weeks dating from the commencement of the catarrhal stage; but it not infrequently lasts longer, even up to twelve or thirteen weeks.

VARIETIES OF THE DISEASE.—The severity of the disease varies. Sometimes it is so mild as almost to escape detection, sometimes it is very severe. The catarrhal stage is occasionally so mild and short that the disease appears to begin with the paroxysmal cough. The “whoop” of the paroxysmal attack may be absent in very young children, and also in severe cases in older children, and then the attacks of coughing may be accompanied by symptoms of laryngeal obstruction. In the cases of whoopless attacks in young children the series of coughs produces extreme cyanosis and suffocation, so that death may occur actually during a paroxysm. In these cases localised muscular spasms, producing squint and

contractions of the toes and fingers, and even generalised convulsions, may sometimes be observed during an attack.

Complications.—Of these the most important are lobular pneumonia and convulsions.

Lobular pneumonia arises during the paroxysmal stage. It should always be suspected if, during the interval between the paroxysms, the patient is ill. The inflammation is usually diffused through both lungs, and, according to Dr. Goodhart, especially affects the anterior edges and roots of the lungs. The usual physical signs of lobular pneumonia are present. Sometimes the consolidated patches are so numerous that the whole of a lobe may appear to be solid. Occasionally there is also *pleurisy*. *Pericarditis* occurs, but is rare. The lobular pneumonia of pertussis is apt to become chronic, and the patches of consolidation may become caseous. *Caseation of the bronchial glands* is not at all an uncommon sequel of whooping cough complicated by lobular pneumonia. Such glands may act as foci of a general tuberculosis. The bronchial catarrh, not often absent from a case of pertussis, may lead to *atelectasis*, *dilated bronchial tubes*, and *emphysema*. Various deformities of the chest, especially transverse constriction and “pigeon-breast,” may occur as the result of deficient expansion of the lungs.

Convulsions are met with mostly in young children. They arise during the paroxysmal stage, and sometimes accompany lobular pneumonia.

The other complications that require mention are *ulceration of the frenum linguæ*, *epistaxis*, *hæmoptysis*, *meningeal hæmorrhage*, and *laryngitis*. The epistaxis and hæmoptysis, though common, are rarely grave.

In severe cases of pertussis marked *emaciation* takes place.

Relapses and **second attacks** are very uncommon.

Duration of Infectivity.—The patient is infectious from the very commencement of the attack, and remains so as long as the characteristic whoop is present.

Morbid Anatomy.—There are no characteristic lesions met with in fatal cases of pertussis. The most common morbid conditions are bronchitis, lobular pneumonia, collapse of the lung, and inflammation of the bronchial glands.

Pathology.—There is every reason to suppose that a micro-organism is the cause of the disease, but up to the present time it has not been isolated. Probably the lungs are the seat of invasion, and the catarrhal stage represents the period of growth of the micro-organism, the paroxysmal attacks being caused by the absorbed toxines, and being comparable to the convulsions in tetanus.

Diagnosis.—There is very little difficulty in diagnosing the nature of the disease when once the paroxysmal cough with the “whoop” has become established, but during the catarrhal stage it may be impossible to arrive at a correct conclusion. Suspicion will be aroused by attacks of coughing, which tend to become paroxysmal, and by the existence of other cases of pertussis in the house or neighbourhood. In the late stage the puffy appearance and ecchymosis of the face are often valuable signs in cases when a history of the illness is lacking.

Prognosis.—Age is of much importance in making a prognosis, for pertussis is a very serious disease in children under one year. Of registered deaths the numbers are greatest during the first year of life, nearly half the total deaths occurring during this year. The number is high during the second year. After that it rapidly decreases. At all ages more deaths occur amongst females than males.

The existence of much bronchitis and lobular pneumonia makes the prognosis more grave ; in fact, these complications are the most frequent cause of death. They arise more often in the winter than the summer season of the year.

Convulsions are very serious.

Treatment.—The child should be kept, during the catarrhal and during the early part of the paroxysmal stage, in a warm but well-ventilated room. Exposure to cold or draughts is to be avoided. The diet should be simple and easy of digestion. It is often advisable to give small quantities of nourishment at frequent intervals.

During the *catarrhal stage* expectorants are useful—*ipecacuanha*, nitrate of potash, acetate of ammonia, etc. Linseed poultices, sprinkled with mustard, should be applied to the chest. The air of the apartment should be kept moist by steam. In a ward a steam tent should be employed. In severe cases brandy in small doses is beneficial.

During the *paroxysmal stage* belladonna in large doses (ten minims of the tincture three times a day for a child of three years, the dose to be increased gradually) is the favourite remedy. At the Evelina Hospital it is given in combination with carbonate of potash. Next to this drug the bromides of ammonium and potassium are the best ; with these chloral hydrate may be combined. Lately hydrochlorate of cocaine, in doses of from $\frac{1}{16}$ to $\frac{1}{4}$ of a grain, according to the age of the patient, three times a day, has been employed with some success. When there is excessive secretion from the bronchial tubes, alum in doses of one to five grains every four hours is of great service in many cases. Compound tincture of camphor, or a small dose of Dover's Powder, given in the evening, is also useful.

During convalescence the patient should take cod-liver oil, and some preparation of iron, such as Parish's Food.

Isolation should be enforced for at least six weeks from the first whoop, for the patient is not to be considered free from infection until the paroxysmal cough has ceased.

CHAPTER XIV.

MUMPS. SPECIFIC PAROTITIS.

MUMPS is a specific fever, of which the chief symptom is inflammation of the salivary glands.

Etiology. Distribution.—It is prevalent all over the world, occurring usually in the form of local epidemics.

Seasonal Prevalence.—It appears to be more prevalent during the cold and wet seasons of the year.

Dissemination.—It is markedly infectious from patient to patient. There is no evidence to show that the infection is carried by a third person or harboured in fomites.

Age and Sex.—It is most commonly met with in children between ten and fifteen years of age, and boys are more liable to be attacked than girls. It occasionally attacks adults.

The **period of incubation**, reckoning from the exposure to infection to the onset of the parotitis, varies from fourteen to twenty-five days. It is commonly three weeks.

Clinical History.—There is occasionally a short prodromal period of one or two days' duration, in which there are symptoms of moderate fever. But usually the disease starts with pain and tenderness in the region of one *parotid gland*. Very quickly the gland becomes swollen, the swelling occupying the space behind the angle of the jaw and below the ear, reaching forward on

to the cheek, and downwards along the neck. The edge is ill defined, and the swelling itself is doughy to the touch. The skin over it may be reddened, but is otherwise not affected. The swelling becomes so extreme and the pain so acute, that the patient can hardly do more than separate the upper and lower teeth. Usually the *submaxillary gland* on the same side becomes affected within a day or two, and there is a large swelling below the jaw. Soon afterwards, the opposite parotid and submaxillary glands become similarly inflamed. The cervical *lymph-glands* may also be involved. Occasionally the inflammation is confined to the submaxillary glands. Dr. Goodhart states that a swelling of the cervical lymphatic glands may be the only local sign of mumps.

With swelling of the parotid and other glands there is malaise and pyrexia. The latter is usually slight, though the temperature may reach 104° Fahr., and there may be delirium. The swelling lasts four or five days, and then subsides, the temperature at the same time falling to normal and the other symptoms abating. Suppuration never results. Usually the amount of saliva secreted is not altered; but it may be excessive, or, on the other hand, diminished. Dr. Goodhart has pointed out that in severe cases where the swelling is marked the patient breathes with open mouth; hence the tongue becomes dry and brown, a symptom which, under these circumstances, is not to be considered of serious import.

Though the salivary glands on each side are usually affected, those on one side very shortly after those on the other, yet the disease may be confined entirely to one side, or the opposite side is not affected until a week or two later.

Complications.—*Orchitis* sometimes occurs. It is met with especially at the age of puberty or in young adults.

It usually arises during convalescence, almost invariably, according to Dr. Clement Dukes, on the eighth day of the disease ; but it may occur early ; very occasionally it is the only local symptom of an attack of mumps. It is accompanied by severe pain and by fever, the temperature frequently reaching 103° or 104° . Often the temperature rises before the testis becomes either swollen or painful. The constitutional symptoms are often alarming ; there may be delirium, and an abnormal slowing of the pulse rate. These symptoms, together with the inflammation of the testicle, subside after a few days. The orchitis may, however, lead to an atrophy of the organ. *Epididymitis* is a rare complication. *Mastitis* is also rare ; it is accompanied by fever and local pain, and may lead to atrophy of the breast. *Inflammation of the ovaries* and *labia* have also been described, but are certainly exceedingly rare complications.

Protection.—One attack usually protects, but as many as three attacks in the same individual have been recorded.

Period of Infectivity.—The patient is especially infectious during the time when the salivary glands are inflamed, and there is reason to believe that he is infectious during the prodromal stage. He should be isolated for three weeks from the onset of the glandular affection.

Morbid Anatomy.—Little seems to be known of the morbid anatomy of this disease. It is stated that the inflammatory process affects rather the connective tissue than the parenchyma of the salivary glands. There is usually some cellulitis around these glands, and the neighbouring lymph-glands are often inflamed. The inflammation never leads to suppuration.

Pathology.—The disease is without doubt caused by a micro-organism ; but this has not yet been discovered. The salivary glands are probably the seat of invasion.

The orchitis may be due to a secondary localisation of the virus.

Diagnosis.—This is usually easy. It should, however, be remembered that there are other conditions which may be accompanied by parotitis. In enteric and other fevers, and in various disorders of the abdominal cavity or viscera, one or both (usually one) parotids may be inflamed. In all these conditions suppuration of the parotid gland may ensue.

Mumps has not infrequently been mistaken for diphtheria; but in this disease the parotid glands are not affected, nor does the patient have any difficulty in opening his mouth, even when the cervical lymph-glands are much enlarged. An examination of the fauces will clear up the nature of any doubtful case.

Prognosis.—This is almost always favourable; for only 80 deaths annually from mumps were registered amongst the entire population of England and Wales during the ten years 1881 to 1890, and probably some of these were really cases of other diseases, such as diphtheria.

The parotitis may result in a chronic inflammation of the parotid glands, and orchitis lead to atrophy of the testis.

Treatment.—There is no special treatment. The patient is able to take nourishment only in the form of liquid or jelly. Pain is relieved by Dover's Powder, and by the local application of hot fomentations with a few drops of laudanum sprinkled upon the lint, or of belladonna and glycerine. Pyrexia and delirium are best treated by sponging with tepid water. Poultices or hot fomentations will relieve the pain and swelling of orchitis. In some cases, however, the application of ice is more beneficial. Dr. Dukes states that this complication never arises if the patient is kept in bed over eight days.

CHAPTER XV.

EPIDEMIC INFLUENZA.

THIS is an acute febrile disease which occurs in well-marked epidemics. Its symptoms are variable. Pyrexia, headache, pain in the back and limbs, are the most constant, and are often accompanied by gastro-intestinal, cerebral, and pulmonary disturbances.

Etiology.—Influenza is a disease which occurs in widely spread epidemics, often separated from one another by long intervals of time. The principal epidemics in England during the present century occurred in 1803, 1833, 1837-8, and 1847-8. The disease re-appeared in England in the winter of 1889-90, after an interval of forty-one years; and this epidemic is noteworthy as one of the most widely spread and most severe that have been recorded. Since this period sporadic cases have been constantly present, and distinct epidemics occurred in the spring and summer of 1891, in the winter of 1891-2, in the spring of 1893, in the winter of 1893-4, and in the spring of 1895. It is not known what becomes of the virus in the intervals between widely separated epidemics, such as, for instance, between the years 1848 and 1889; and there is no distinct evidence of the occurrence of sporadic cases during this interval.

When the epidemic of 1889 visited England influenza

was practically a new disease to most medical men in the country, although there can be no doubt that it was the same disease as the one which was epidemic in the years above mentioned. At first it was considered to be a disease prevalent in hot climates known as Dengué; but the two diseases differ so much from one another in clinical aspect, geographical distribution, and climatic prevalence, that their non-identity can be considered established.

Epidemics of influenza are widespread. Race, climate, and season have no apparent influence over their distribution. The population is attacked without distinction of age, sex, constitution, or condition. An epidemic develops with such rapidity that the disease is supposed by many to be caused by some atmospheric condition, and not to spread directly or indirectly from one individual to another. It has even been denied that the disease is infectious. But Dr. Franklin Parsons, in a very careful report on the epidemic of 1889-90, came to the conclusion that the disease spreads from individual to individual, either directly or by means of fomites, for the following reasons. The epidemic in the northern hemisphere spread from east to west, contrary to the direction of the prevailing winds. It appeared to follow in the main the lines of human intercourse, the large cities being attacked before the smaller towns and rural districts. It did not travel faster than human beings, parcels, and letters could travel. In most districts the epidemic was preceded by a few scattered cases. Lastly, in institutions in which the inmates were brought closely into association, the disease prevailed more extensively than in those in which the inmates were more secluded from one another.

The **incubation period** is certainly short, probably two or three days, sometimes less.

Clinical History.—The disease generally commences quite suddenly, many patients being attacked while at their usual occupation. The symptoms at the onset are slight shivering, malaise, severe frontal headache, and pains in the back and limbs, followed by prostration. Vomiting, fainting, or giddiness is sometimes the first symptom.

In mild cases the attack begins insidiously; and it is then difficult to assign the date of onset.

TYPES OF THE DISEASE.—The disease rapidly develops after the onset, and then assumes one of several types. These are the febrile, the bronchitic, the catarrhal, the gastro-intestinal, and the cerebral.

Febrile type.—This was the most common type during the epidemic of 1889-90.

After the onset the usual symptoms of fever follow, such as loss of appetite, furred tongue, and constipation. The temperature rapidly rises to 102° or 103° , though it may reach 105° or 106° . The respirations are often much increased, although there is no evidence of bronchitis. There may be a troublesome cough, but there is no expectoration. The patient always feels very ill, and often complains of giddiness, restlessness, and an entire inability to concentrate his powers of thought. Pains in the back and limbs, and frontal headache are prominent symptoms, and are often very severe. The pain may affect almost any part of the body, and the joints are not especially attacked. Profuse and persistent perspiration is very common. The pulse is quickened in correspondence with the temperature. In some cases it becomes very rapid and irregular. Herpes may appear upon the face.

The duration of the disease, when no complication ensues, is short. After two or three days the temperature falls, and with it the other symptoms subside.

Convalescence is prolonged, and it is some time before the patient regains his normal health and vigour. Loss of weight, great muscular weakness, a tendency to break out into profuse perspiration, a lack of mental energy, and depression of spirits, are the principal symptoms observed during convalescence.

Bronchitic type.—This form of the disease is the most fatal. Shortly after the usual symptoms have appeared the breathing is found to be rapid and difficult, there is a sense of constriction and pain in the chest, and a cough develops. The expectoration either consists of glairy mucus, sometimes tinged with blood, or of small yellowish-green pellets. Auscultation reveals the presences of râles and rhonchi. Not infrequently the inflammation spreads from the bronchi to the pulmonary tissue, and a form of pneumonia results. The consolidation is not, as a rule, limited to one lobe, as in fibrinous pneumonia; it has more the type of a lobular pneumonia, although by a confluence of various patches a large area of lung may be involved. A true croupous or lobar pneumonia may, however, occur as a complication. In severe cases the temperature is high, and there is sometimes hyperpyrexia. The pulse is often extremely rapid and weak. Delirium is not uncommon. In fatal cases the patient becomes cyanosed, the difficulty in breathing increases, and the heart fails.

Catarrhal type.—In some epidemics this is the most common type. The symptoms, in addition to those of the febrile type, are pain of a burning character in the eyes, forehead, and nose. Sore throat and cough are frequently complained of. A discharge of a thin acrid

fluid, which rapidly becomes purulent, flows from the nose. The mucous membrane of the pharynx and nasal cavities is seen to be reddened and inflamed, and there may be a pultaceous exudation upon the tonsils. The catarrh lasts several days, and then either disappears, or spreads to the trachea and lungs, in which event the case assumes the bronchitic type.

Gastro-intestinal type.—In this form, which is uncommon, the prominent symptoms are those of an inflammation of the gastro-intestinal tract. In addition to the symptoms of the febrile type there is severe abdominal pain, diarrhœa, and vomiting.

Cerebral type.—In this form, which is also uncommon, the most marked symptoms are pyrexia, delirium, stupor, and sleeplessness. Retraction of the neck, squint, and other symptoms simulating meningitis have been observed.

Mild cases.—It must not be understood that every case conforms to one of the types described. Cases frequently occur of a mixed type. The cerebral and bronchitic symptoms, for instance, may both be so prominent that the case might be described equally well under the heading of either the cerebral or bronchitic type. But besides these mixed cases there are mild forms in which many of the characteristic symptoms are absent. Pyrexia is frequently absent, while the pains, the mental depression, and the prostration are well marked. In other cases only one symptom, such as persistent giddiness, general malaise, mental depression, or bronchitis, may constitute the attack. Such cases may begin suddenly ; but they may be insidious in their onset and course, and are often unrecognised. Even in these mild cases convalescence is slow, and for some time the patient suffers from muscular weakness and lack of mental energy.

Complications.—Various *suppurative* affections by no means uncommonly occur during an attack of influenza. We will especially mention *acute suppurative otitis*, *suppuration in the antrum of Highmore*, and *acute parotitis*.

Pleurisy and *empyema*, *albuminuria* and *herpes*, are also sometimes met with.

Various *rashes* of a scarlatinal, measly, or petechial character have been described; but they are rare.

Hæmorrhages sometimes occur from the lungs, nose, vagina, or bowels.

The *heart* may become dilated, the apex moving outwards, and the pulse becoming rapid and irregular. A fatal termination from this cause is not uncommon.

In addition to the pains already described, various forms of *neuralgia* may either complicate or follow upon an attack of influenza. Sometimes the pain is in the chest, and simulates pleurisy or angina pectoris, sometimes it affects other parts, such as the rectum. The pain is often agonising, the patient being unable to move during a paroxysm.

Sequelæ.—During convalescence *bronchitis* and *pneumonia* are frequent. The *pneumonia* of convalescence should be distinguished from that occurring during the acute stage. It is of the usual-lobar type, and only differs from a primary attack of acute lobar pneumonia in being more fatal. *Phthisis* is not an uncommon sequela; and in tuberculous patients an exacerbation of the disease frequently follows on influenza.

Mental depression is fairly constant, and forms one of the most characteristic features of the malady. *Insanity* may arise from an attack of influenza in patients who are predisposed by heredity. The disease usually assumes a

melancholic type, but mania and other forms have been described. *Peripheral neuritis* sometimes occurs as a sequela.

An *irregular* heart, with or without definite signs of dilatation, by no means infrequently remains after an attack. Frequent attacks of fainting are not uncommon. *Giddiness, muscular weakness, and lack of mental vigour* may persist for a long time.

Relapses and Second Attacks.—The protection afforded by one attack is slight and transient. Many persons are especially susceptible to influenza, and suffer during every epidemic. It is quite common to meet with patients who have had three, four, or more attacks. Relapses are also frequent. Just as the patient has recovered from one attack another begins, and is often of a different type from the first.

Length of Infectivity.—The disease is infectious in the early stage, but the length of infectivity varies in individual cases.

Morbid Anatomy.—In some cases of influenza nothing of importance is found at the post-mortem examination; but in the majority of cases the respiratory tract is affected. The mucous membrane of the trachea and bronchi are intensely congested, and the tubes filled with muco-pus.

Pneumonia is frequently present. Sometimes, especially if it has occurred during convalescence, there is nothing to distinguish it either macroscopically or microscopically from an ordinary lobar pneumonia. But in other cases it is of a special type. A large area—a whole lobe, for instance—may be affected, although a careful examination shows that this is due to a confluence of many patches of lobular pneumonia. A microscopical examination reveals an abundant cell infiltration with little or no fibrinous

exudation. More rarely patches of gangrene or suppuration can be seen. When death has occurred from one of the complications already described the corresponding lesions will be found.

The *influenza bacillus* is very minute, and is two or three times as long as broad ; but it sometimes grows out to form short threads. The ends of the bacillus are frequently more darkly stained than the centre, giving the appearance of a diplococcus.

The best method of obtaining cultivations is to sow some of the bronchial secretion, which has been washed in sterilised water, upon the surface of agar streaked with pigeon's blood. The tubes are placed in the incubator at 37° C. ; and at the end of twenty-four hours very minute, almost microscopical, colonies develop. The cultivations very rapidly lose their vitality, and must be transplanted from day to day.

Diagnostic value of a bacteriological examination.—The discovery of the influenza bacillus in the sputum is a valuable aid to diagnosis. The method of preparing specimens is as follows :—The sputum, directly after being coughed up from the bronchi, is washed in sterilised water, and from the interior of the mass cover-glass preparations are made. The cover-glass is then stained in a dilute carbolic fuchsin solution, and is mounted in the usual way.

Pathology.—The influenza bacillus is probably the cause of influenza. It can be found in a large majority of the cases of influenza, and has not been found in any other disease. It is chiefly the bronchial type of the disease that has been investigated. The bacillus is found in the bronchial mucus, and in sections it is seen to penetrate into the submucous tissues of the bronchioles. In the secretions from the trachea and nose the bacillus is more difficult to

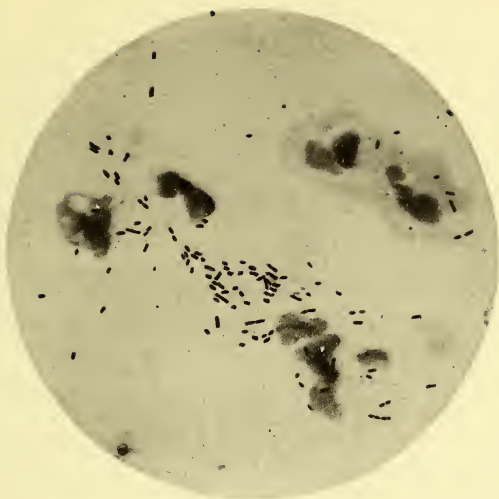


Fig. 1.—The Influenza bacillus in sputum. $\times 1000$.

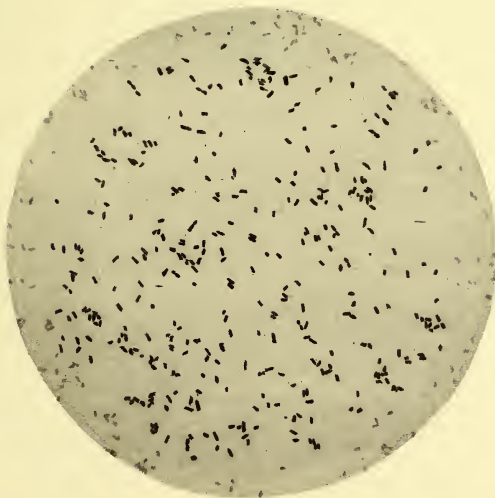


Fig. 2.—The Influenza bacillus. Cultivation $\times 1000$.

discover, on account of the presence of other bacteria. It has sometimes been found in the organs, and rarely in the blood. In the pneumonia occurring during the acute stage of the disease the bacillus is present, sometimes alone, and at other times in association with a streptococcus, the pneumococcus, or other bacteria. When influenza bacilli are alone present the inflammation of the lung is characterised by the absence of fibrin in the exudation. The pneumonia following on influenza is generally due to the pneumococcus, and is similar to ordinary acute lobar pneumonia.

In view of the fact that the bacillus does not multiply in the blood or organs, we must consider that influenza is a local disease, usually of the respiratory tract, and that the symptoms are due to the absorption of the toxins.

Experiments upon animals give us but little information about the pathology of the disease, for most animals are not susceptible to inoculation. Pfeiffer has produced the symptoms of influenza by inoculating monkeys, but others have failed.

An important point in connection with the etiology of the disease is the fact that the bacillus is sometimes present for weeks or months in the secretions of convalescent patients. It is improbable that the bacillus multiplies outside the body in the soil or in water.

Relation of influenza to epidemics among animals.—During an epidemic of influenza domestic animals, such as dogs, cats, and caged birds, are frequently affected; and it is probable, though not proved, that the disease is the same. An infectious disease has also been observed to be prevalent among horses at the same time as an epidemic of influenza, but there is not sufficient evidence to show

that the two diseases are connected. "Horse Influenza" probably includes several distinct diseases, and some forms occur at times when the human race is not affected.

Diagnosis.—A typical case of the febrile type with severe pains is easy to diagnose, and is only likely to be mistaken for rheumatism. The absence of swelling of the joints and the sudden onset are sufficient to prevent an error in diagnosis.

When the pains are slight or absent greater difficulties arise, and the disease may be mistaken for various febrile conditions. A sudden onset, attended with severe prostration and mental depression, is characteristic.

Severe cases of the bronchial type are liable to be mistaken for commencing lobar pneumonia. The sudden onset, pain, herpes, signs of consolidation, pyrexia, and albuminuria, when present, render the diagnosis difficult. In influenza there is generally bronchitis all over the lungs in addition to the consolidation; and the latter is more indefinite, and is not limited to one of the lobes, as in pneumonia. A true lobar pneumonia does, however, frequently complicate influenza, but usually occurs at a late period, and should be reckoned as a sequela rather than a complication. Should the two diseases occur simultaneously the pneumonic symptoms quite mask those of the influenza, and it is difficult to say whether the patient is suffering from the latter disease or not. During epidemics of influenza there is an increase in the number of cases of pneumonia, and this is probably due to the former disease predisposing patients to the latter.

Mild cases, especially when unattended with fever, are difficult to recognise.

The gastro-intestinal form may be mistaken for gastro-enteritis, colic, enteric fever, and intestinal obstruction.

The cerebral form may be mistaken for meningitis, cerebral abscess, etc.

The catarrhal form is often difficult to diagnose from a severe cold.

In all cases severe pains, mental depression, and tedious recovery are valuable signs. The greatest difficulty arises in sporadic cases.

Prognosis.—We have no accurate statistics of the case-mortality of influenza. It is certainly low ; but on account of the large number of persons attacked the general death-rate during an epidemic is greatly increased, principally through the indirect effects of the disease.

The prognosis is generally favourable. The age of the patient is an important factor. The mortality is much higher among elderly patients than adults, and children take the disease in a mild form.

In individual cases high fever, severe cerebral symptoms, evidence of extensive implication of the lungs, and cardiac failure are of serious import. Patients whose constitutions are broken down by alcoholic excess or by chronic disease are especially bad subjects.

Of the complications pneumonia is the most grave, the mortality being exceedingly high.

In tuberculous patients the tuberculous process is apt to rapidly increase.

Treatment.—Patients suffering from influenza should always keep their beds until convalescence is well established, and they should remain indoors for some days later. Nothing is more dangerous than to attempt to ignore the disease, and to continue the ordinary occupations of life. A fatal termination is often due to the sequelæ of mild and neglected cases.

The diet during the acute stage should consist of milk,

beef-tea, chicken broth, peptonised food, bread, jelly, etc. A few days after the fever has subsided fish and poultry may be added.

Pyrexia should be treated with tepid or cold water sponging, or wet packs. Salicylate of soda or quinine are useful in febrile cases. The former drug will also relieve the pains in the back and limbs. For the headache anti-pyrin in small doses is very beneficial. The pain in the back is much relieved by such applications as a mustard poultice.

In the bronchitic type of the disease stimulating expectorants should be administered.

When there is cerebral excitement the best drug to employ is morphia by subcutaneous injection.

Complications should be treated on general principles. During convalescence tonics are indicated. Alcohol is beneficial in cases where the heart shows signs of failure. It is especially of use in severe febrile cases, in the bronchitic form of the disease, and in those patients who are accustomed to take it as part of their diet.

The convalescent should be particularly warned against running any risk of "catching cold" for some time afterwards. He should avoid cold winds and draughts, and as much as possible sudden changes of temperature, and should be warmly clad.

On account of the extremely infectious nature of the disease the spread of an epidemic cannot be prevented by isolation. Nevertheless, in institutions such as lunatic asylums and boarding schools, it has sometimes been possible to keep the disease from spreading by prompt and careful isolation of all suspicious cases.

CHAPTER XVI.

TYPHUS FEVER.

TYPHUS FEVER is an acute infectious disease, characterised by a distinctive eruption, and attended by severe constitutional symptoms. The fever lasts about a fortnight, and terminates by crisis. A number of names have been given to this disease, such as gaol fever, spotted fever, camp fever, etc.

Etiology.—It is only within the last fifty years that typhus has been differentiated from enteric fever. Formerly typhus and enteric, together with certain other fevers, were all included under the name of continued fever.

Geographical Distribution.—In spite of the former confusion between these fevers there can be no doubt that typhus was at one time very prevalent all over Europe. It has also been prevalent in China, Persia, Egypt, and America. Australia, New Zealand, Japan, and the greater part of Africa appear to have escaped. In Great Britain it was especially prevalent in Ireland, occurring in the form of epidemics every few years, but at the same time being more or less endemic. During the past few years it has decreased very considerably, and now occurs only in small localised outbreaks.

Seasonal Prevalence.—In England the disease has been

more prevalent, and the mortality higher, during the winter and spring.

Dissemination.—Typhus is exceedingly infectious from person to person, and it is in this way that the disease is usually disseminated. The virus is given off with the breath, and rapidly loses its potency when diluted with air. Infection may also be harboured in fomites, and the disease may thus be spread should proper precautions not be taken.

There can be no doubt that starvation and overcrowding are important factors in the production of an epidemic, but this must not be considered as a proof of the *de novo* origin of typhus. Badly fed individuals are more susceptible to the disease; and should the virus be introduced into an overcrowded and poverty-stricken community an extensive epidemic will almost certainly arise. It is for these reasons that military camps and the populations of besieged cities have in former years suffered so severely.

Table showing admissions, deaths, and case-mortality of the patients suffering from typhus fever admitted into the hospitals of the Metropolitan Asylums Board from 1871 to 1894:—

Ages.	Cases admitted.	Died.	Case-mortality. Per cent.
Under 5	88	2	2·3
5 to 10	245	1	0·4
10 „ 15	377	15	4·0
15 „ 20	360	28	7·8
20 „ 25	247	50	20·2
25 „ 30	158	36	22·8
30 „ 35	161	47	29·2
35 „ 40	133	47	35·3
40 „ 45	170	81	47·7
45 „ 50	96	42	43·8
50 „ 55	61	37	60·7
55 „ 60	32	24	75·0
and upwards	38	27	71·1
Totals	<u>2166</u>	<u>437</u>	<u>20·2</u>

Age and Sex.—From the foregoing table it appears that typhus is not common in children under five years of age. The numbers increase during the next quinquennia, and reach a maximum in the period from fifteen to nineteen years. After this age the number of cases gradually decreases. Sex has no influence on its incidence.

The **incubation period** is somewhat variable. As a rule, it is twelve days; occasionally it is longer; not infrequently it is shorter, from eight to ten days, and it may be as short as five days.

Clinical History.—The attack usually begins suddenly with a slight rigor, or a feeling of chilliness, accompanied by frontal headache and pains in the limbs. The temperature rises during the first day of the illness, and on the second day is 102° or 103° . Between the fourth and the seventh day a maximum of 104° , 105° , or even 106° Fahr. may be reached. As the temperature rises there is an increase in the respiration and pulse rate. The tongue becomes furred, the appetite is lost, and the bowels are confined. The face becomes flushed, the conjunctivæ injected, and the countenance assumes a dull and vacant expression. Vertigo and buzzing in the ears are nearly always present, and there is sleeplessness and slight delirium at night. Towards the end of the first week deafness often sets in. Prostration is a marked symptom, and by the third day the patient usually takes to his bed. A peculiar and offensive odour is often to be observed.

The *eruption* makes its appearance on the fourth or fifth day of the disease, but it may be delayed until the sixth, seventh, or eighth day. It is first seen on the anterior axillary folds, and the sides of the abdomen, or upon the backs of the hands and wrists; then it spreads to the rest of the trunk, the arms, and the lower ex-

tremities. It very rarely affects the neck or face, but the latter is deeply flushed. At first the rash consists of minute



DIAGRAM XII.—TYPHUS FEVER.

An ill-defined papular, blotchy, and petechial rash ; seen best upon the sides of the chest and abdomen, the wrists and ankles. The face is usually unaffected.

dusky-red spots, which are very slightly elevated ; they are smaller than those of enteric fever, and the edge is

more indefinite, the redness gradually fading into that of the surrounding skin. The spots occur usually in considerable numbers, and are often arranged in small



DIAGRAM XIII.—TYPHUS FEVER.

clusters. Some of the spots may coalesce so as to form larger irregularly shaped blotches.

In addition to the spots already described there are

many paler in hue and with a still more indefinite outline. They give the impression of spots seen through the cuticle ; and, when looked at from a distance, the skin has a mottled appearance. This is known as the "subcuticular mottling."

Fresh spots continue to come out for two or three days, but after the third day no more appear. At first the eruption disappears on pressure, but after a day or two it becomes of a darker and more purple colour, and then only fades when pressure is applied. Many of the spots now become distinctly petechial, especially in the groins, bends of the elbows, and on the back. The hæmorrhage is usually limited to the centre, but the whole of the spot may be involved. At this stage the colour of the eruption is like that of mulberry juice. Hence the term "mulberry rash." After the eruption has lasted several days it begins to fade, but does not entirely disappear before the fourteenth day of the disease. Petechiæ often persist longer, even up to the twenty-first day.

Sometimes the rash is absent, and sometimes it is limited to the parts upon which it first appeared. The spots may never become petechial, stopping short at the stage at which they disappear on pressure. The relative proportion between the spots and the subcuticular rash is variable, and one or the other may be absent.

Course of the disease.—From the onset of the attack until the end of the second week the patient gradually gets worse. Shortly after the rash has appeared the temperature falls slightly, but remains at a high level, 103° to 105° Fahr., with morning remissions and evening exacerbations of one or two degrees. The tongue becomes dry and brown, and in severe cases black and contracted ;

the lips and teeth are covered with sordes and the breath is offensive. Constipation is usually pronounced. The pulse increases in frequency, and becomes compressible. The respirations also increase, and an examination of the chest reveals the presence of hypostatic congestion of the bases of the lungs. Deficient aeration of the blood is shown by a duskiness of the face and a lividity of the lips.

Cerebral symptoms are marked. The headache, which was present during the prodromal stage, disappears; but sleeplessness remains, and delirium becomes a prominent feature. The delirium assumes the character of a low muttering type; occasionally the patient is violent, and requires careful watching to prevent him from doing himself harm. In many cases the patient is quite unconscious, or can only be roused by shouting loudly in his ear. The pupils are minutely contracted. In severe and generally fatal cases a condition known as "coma vigil" supervenes. In this condition the eyes remain open, yet the patient is quite insensible to what is going on around him.

Muscular prostration is a marked feature of the disease, and the patient lies helplessly on his back in the bed. Muscular tremor and subsultus tendinum are pronounced. In severe cases the urine and fæces are passed involuntarily, but retention of urine is not uncommon.

In fatal cases the pulse becomes exceedingly rapid and weak, and the hypostatic congestion of the lungs increases. Death generally occurs at the end of the second week, and is preceded for some time by absolute coma. Sometimes death takes place during the third week, the temperature having remained high during the whole period of the disease.

In cases which recover the temperature falls by crisis,

generally on the thirteenth or fourteenth day. With the fall of the temperature the other symptoms rapidly subside, and the patient soon regains his normal health.

VARIETIES OF THE DISEASE.—There are *malignant* forms of the disease (typhus siderans, or blasting typhus), in which the patient dies on the third or fourth day.

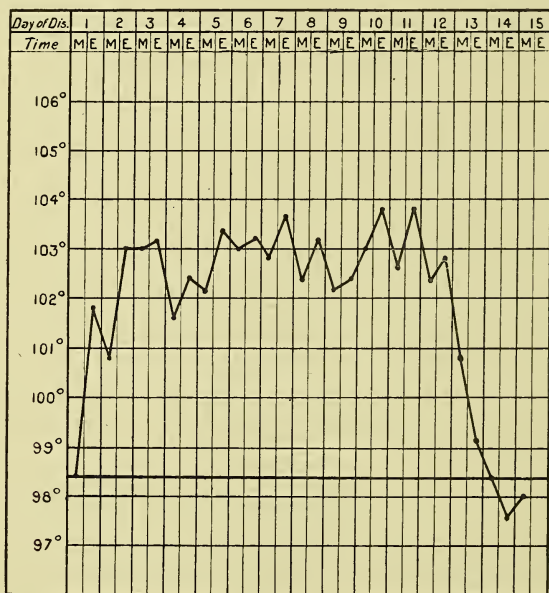


CHART T.

Moderately severe attack of typhus fever in a female aged 27 (after Copeman).

There are also *mild* cases, in which the symptoms are mild throughout, and in which the crisis may occur as early as the eighth day. In these cases the rash may be ill-defined.

Complications.—There are not many complications in typhus.

Lobar pneumonia is not infrequent, and is often insidious

in its onset. The affected lung may become gangrenous. *Pleurisy* with or without effusion may occur, and it is important to remember that both pleurisy and pneumonia may come on after the crisis. *Laryngitis* is a rare complication. The muscle of the *heart* may become degenerated, but *endocarditis* and *pericarditis* are very rare. Cases are recorded in which cardiac *thrombosis* and subsequent *embolism* have been observed. *Albuminuria* is common, but does not necessarily imply any serious renal lesion. *Acute nephritis* has been observed, but is not common. Retention of urine may lead to *cystitis*. *Inflammatory swellings*, leading to suppuration, are occasionally met with in the submaxillary and parotid regions. *Convulsions* may set in towards the end of the second week. *Mania* occasionally results from an attack of typhus, and may necessitate the removal of the patient to an asylum. Recovery usually takes place in a few months.

Other *rare complications* are gangrene of the toes, fingers, penis, or scrotum, cancrum oris, purpura hæmorrhagica, paraplegia, and peritonitis.

Protection.—Relapses are excessively rare, and second attacks very uncommon.

Length of Infectivity.—The patient is free from infection at the end of four weeks from the commencement of his illness.

Morbid Anatomy.—There is no constant or distinctive morbid appearance, either macroscopic or microscopic, met with post mortem in typhus fever. The petechiæ, and even the mottling, may be still visible on the skin. Hypostatic congestion of dependant parts is well marked. Decomposition rapidly sets in. The blood is usually fluid, or but slightly clotted. The spleen may be enlarged, and is friable. The lungs, especially their bases and

posterior parts, are engorged with blood. There may be slight degenerative changes in the heart muscle, and cloudy swelling of the hepatic and renal cells.

Pathology.—Micro-organisms have been found in the blood, but there is not sufficient evidence that they are the cause of the disease.

Diagnosis.—Typhus is most often confounded with enteric fever, pneumonia, purpura, measles, acute tuberculosis, and tuberculous and other forms of meningitis. It may also be confused with small-pox and relapsing fever.

The onset of *enteric fever* is in most instances slow and insidious, while the contrary is the case in typhus. In the latter disease the patient quickly becomes prostrate, but not in the former. Headache is a very prominent symptom of the initial stage of enteric fever, and in this disease the temperature often rises in a zigzag manner during the first three or four days of the illness. The rash of enteric fever consists of definite spots, that of typhus of indefinite spots and mottlings, together with petechiæ. In typhus fever the spleen is not as a rule enlarged, diarrhœa is not a common symptom, and the disease generally terminates by crisis. In enteric fever the pupils are dilated and in typhus contracted.

Pneumonia is to be distinguished by the rapid breathing, cough, rusty sputum, labial herpes, and physical signs of lung-consolidation and pleurisy. Even in cases where these physical signs are wanting, the increased respiratory rate, cough, and herpes will indicate the nature of the illness.

Purpura is not infrequently mistaken for typhus on account of the eruption. But the eruption in purpura is hæmorrhagic from the beginning, and consists of

well-defined red or purple spots, and larger bruise-like subcutaneous hæmorrhages; it has not the mottled appearance of typhus fever. The course of purpura is usually slower than that of typhus, and, during the first few days at any rate, the febrile symptoms are not marked. In the severe form there is hæmorrhage from one or more of the mucous membranes.

A purpuric eruption sometimes occurs in nephritis, ulcerative endocarditis, and other diseases.

The rash of typhus is not infrequently very like that of *measles*, especially in the early stage. In typhus, however, the rash is rarely seen upon the face, while it is rarely absent from the face in measles. In the latter disease there is catarrh of the air-passages, with frequent coughing and sneezing.

The onset of *acute tuberculosis* and *tuberculous meningitis* is more gradual than that of typhus. The rapid breathing, and perhaps physical signs of lung mischief in the one case, and the signs pointing to a cerebral lesion in the other, afford indications of the nature of the illness. There is no rash in these diseases. Where some other form of meningitis is suspected search should be made for the cause, such as ear-disease. Optic neuritis, while common in all varieties of meningitis, does not occur in typhus. It should be remembered that in epidemic cerebro-spinal meningitis there may be an eruption, either herpetic (especially on the face), urticarial, or purpuric.

For *small-pox* typhus is only liable to be mistaken during the initial period. In typhus there is no lumbar pain. The third day will usually clear the case up by the outcome of the distinctive eruption in variola.

In *relapsing fever* a rash is very exceptional. The first attack ends by crisis at the end of a week; at the end

of another week the relapse comes on, and lasts three or four days, terminating by crisis. During the primary attack and the relapse a characteristic and diagnostic organism, the *Spirochæta Obermeieri*, can be seen by a microscopical examination of the blood.

Prognosis.—From the Table on p. 266 it appears that children do better than adults, and that the case mortality rises considerably after the twentieth year, and continues to rise with advancing years.

Of 2,166 patients referred to in the Table, 981 were males, and 1,185 females. Of the former 220 died, a percentage fatality of 22·4; of the latter 217, a percentage fatality of 18·3.

The prognosis is grave in patients who have led intemperate lives, and in those who have been deprived of sufficient food and proper housing. The existence of any chronic malady renders the prognosis bad. Pregnancy does not seem to be an unfavourable complication. Amongst symptoms of evil augury arising from the attack itself the following may be especially mentioned: a persistently frequent pulse of over 120 per minute, signs of cardiac failure, frequency of respiration, pronounced nervous symptoms during the first week, coma vigil, extreme prostration, convulsions, the involuntary passing of urine and fæces before the middle of the second week, much cyanosis, profuse sweating a day or two before the crisis is expected, the failure of the crisis to take place, and the occurrence of any serious complication. It is stated by Murchison that a rise instead of a fall of temperature at the end of the first week is an unfavourable symptom.

Treatment.—Typhus is a disease which has, in certain instances, been abolished from communities by paying proper attention to the conditions of life of the lower

classes of the population. Overcrowding and want of ventilation, dirt, and a bad and insufficient food supply, are the conditions with which its presence and spread are most associated. When defects of this nature are remedied, and isolation adopted, typhus can either be kept away altogether, or, if it gain an entrance, limited to a few cases.

With respect to the treatment of the disease itself, the patient should be placed in a well-ventilated room or ward. The windows adjoining his bed should be kept open (except in very cold weather), so that fresh air may circulate freely around him. Not only will the patient receive benefit from such treatment, but the risk of catching his complaint, that those who attend upon him most undoubtedly run, is very much diminished; for it seems that the specific virus loses most of its potency when freely diluted with air.

The diet during the febrile stage is such as is recommended for enteric fever; but three or four days after the crisis has occurred the patient may be allowed fish or poultry, and in another three or four days ordinary diet.

Convalescence is usually steady and rapid, and the patient is soon able to get up.

Sponging with tepid or cold water, or the employment of the wet pack, is beneficial when pyrexia and delirium are marked. One or two doses of quinine (10 to 20 grains) may be given. A mixture containing dilute hydrochloric acid and tincture of orange was recommended by Murchison. In cases where the pulse is very frequent and soft, and there is much prostration, digitalis and strychnine in combination may be prescribed with advantage. Stimulants are also called for in such cases. Ether, ammonia, camphor, and alcohol are the stimulants most frequently used. Of alcohol, Murchison wrote that

patients under twenty years of age did best without it, those over forty were benefited by it, and those of intemperate habits required it earlier and in larger quantities than others. A hot, dry skin, active delirium, and scanty, albuminous urine contra-indicate the administration of alcohol.

The severe headache of the early stage is relieved by the application of cold water to the head, or by leeching the temples. For sleeplessness opium in small doses may be given, but this remedy should be employed with caution towards the middle of the second week, when prostration is pronounced. Chloral hydrate, in combination with bromide of potassium, is the best drug for delirium. For stupor Murchison recommended a cup of strong coffee every three or four hours. When the urine is scanty, dry cupping or mustard poultices to the loins are worthy of trial.

Complications must be treated as they arise, according to their nature. During convalescence some preparation of iron should be administered.

The patient should be isolated for at least four weeks from the commencement of the attack.

CHAPTER XVII.

RELAPSING FEVER. FAMINE FEVER.

THIS is an infectious disease, consisting of an acute febrile attack lasting about a week, and followed at the end of another week by a second similar attack. A characteristic micro-organism is present in the blood during the primary attack and the relapse.

Etiology.—*Geographical distribution.*—During the first half of the present century relapsing fever was frequently prevalent in Ireland and Scotland, less often in England. Its last appearance in epidemic form in Great Britain was between the years 1868 and 1873. On the Continent the disease has been chiefly confined to Russia and Germany; in the latter country it was prevalent as late as the years 1878 and 1879. It has also appeared in various parts of North America, India, China, and Egypt, but not in Australia.

Dissemination.—Relapsing fever is chiefly spread directly from the sick; but the infection may be harboured in fomites. The chief condition that favours its development is starvation, and its prevalence has been especially associated with famines. To a minor degree it depends, like typhus fever, upon overcrowding and squalor. Indeed typhus and relapsing fever have not infrequently been

epidemic in the same place and at the same time ; and some writers believe that an intimate relationship exists between the two diseases.

Season.—The prevalence is greater during the winter than the other seasons of the year.

Age.—The following table shows the percentage of cases occurring at different age-periods amongst 2,111 cases recorded by Murchison :—

Age.	Percentage of total cases.	Age.	Percentage of total cases.
Under 5	1·8.	35 to 40	6·8.
5 to 10	5·9.	40 to 45	6·7.
10 to 15	11·0.	45 to 50	4·2.
15 to 20	19·1.	50 to 55	3·7.
20 to 25	16·8.	55 to 60	1·8.
25 to 30	9·8.	60 and over	2·3.
30 to 35	8·4.		

From this table it will be seen that there are few cases under five years of age, and that the largest number occur between ten and twenty-five.

The average *fatality* is about 4 per cent. It is low under thirty, but increases with advancing years.

Sex.—More males than females are attacked, but the fatality is greater among the latter than the former.

Incubation Period.—This appears to be very variable. Murchison states that it may be one to fourteen days ; other English medical men say four to ten ; the Silesian observers gave seven to twenty-one days as the extremes. Dr. Vandyke Carter found, in his experiments on apes, that the period varied between thirty hours and five days.

Clinical History.—The attack begins very suddenly with marked rigors or slighter shiverings. Soon succeed intense headache, pains in the back and limbs, and giddiness. Sometimes there is also nausea or vomiting. The

temperature rapidly rises, so that in a few hours it reaches 104° , 106° , or even 108° Fahr. At the same time the skin becomes hot and dry, the tongue furred, and the pulse-rate runs up to 120 or 140 per minute. As the disease advances, the pulse-rate increases, the tongue becomes dry, intense thirst is complained of, the bowels are constipated, and there may be vomiting. There is enlargement of the liver and spleen, and pain on pressure over the hepatic, splenic, and epigastric regions. In many cases jaundice appears about the third day. The temperature remains elevated, 103° to 106° , or even higher, with slight remissions, and the pulse keeps at 140 to 160, being often full and firm. The pains in the back and limbs continue, and become much more severe, and there is usually sleeplessness. The urine is not as a rule diminished in quantity. It is occasionally albuminous, and sometimes bile-stained.

At the end of a week, but sometimes as early as the third or as late as the tenth day, the attack suddenly terminates by crisis. Occasionally just before the crisis there is a brief period of delirium or maniacal excitement. The crisis usually commences with profuse sweating, rarely with diarrhœa, epistaxis, hæmorrhage from the bowels, or the menstrual flow. Within a few hours the temperature falls to subnormal, and the pulse-rate drops to below 70. There is not infrequently weakness and irregularity of the heart's action. The tongue becomes clean, the skin cool and moist, the pains cease, and the patient, with the exception of a sense of exhaustion, feels perfectly well.

During the next few days the patient rapidly gains in strength, so that before the end of the week he may be up, and even engaged in his usual occupation.

In the large majority of cases, usually on the seventh day from the crisis (fourteenth from beginning of illness),

the *relapse* occurs. The manner of onset is similar to that which is observed in the primary attack ; so also are the symptoms, which may, however, be more, or less, severe. The relapse is usually of three days' duration, and terminates by crisis. Occasionally it lasts only one or two days, or as long as four or five.

In some instances on the twenty-first day from the commencement of the illness a second relapse follows, and in rare cases there is a third, or even a fourth.

Recovery from the relapse is usually tedious. The spleen may remain enlarged and tender for several weeks, and there may be fever of a remittent type.

In most cases of relapsing fever there is no rash. But small rose spots, or a reddish mottling very like the early stage of the typhus eruption, may exceptionally appear upon the trunk. These rashes are transitory, and they do not become petechial. In a few cases, especially when there is jaundice, there is a purpuric eruption, consisting of petechiæ and purpuric spots. During the critical perspiration sudamina often appear.

In fatal cases death occurs from collapse, suppression of urine, and convulsions, or from some complication such as pneumonia. Collapse is usually met with about the time of the crisis, but it may come on during the period of remission or during the relapse. It is met with in mild as well as in severe cases. The patient, who up to the onset of the symptoms of collapse may have been doing well, becomes cold and livid, the pulse fails, unconsciousness supervenes, and death ensues within a few hours.

VARIETIES OF RELAPSING FEVER.—There is a form of relapsing fever which has been described by some authors under the name of "bilious fever." In these cases

there is nearly always jaundice ; hæmorrhages occur both into the skin and from the mucous membranes ; prostration is a marked feature ; and the patient falls into the "typhoid state." The crisis is absent or imperfect, and the primary attack is prolonged, as it were, into the relapse. The case-mortality is high. That "bilious fever" is a variety of relapsing fever is proved by the presence of the characteristic spirillum.

Complications.—During the acute stages of the disease (the primary attack or relapse), *lobar pneumonia* is not uncommon. *Bronchitis* and *laryngitis* also occur. *Hæmorrhages* into the skin or from the mucous surfaces are not infrequently met with, especially about the critical period, and they may prove fatal. The *spleen* occasionally becomes the seat of abscesses. *Peritonitis* occurs in rare cases.

Collapse, as a fatal event, has already been mentioned ; but it does not necessarily terminate in death. It may occur during the primary attack, remission, or relapse, and in mild as well as in severe cases.

Attacks of diarrhœa are common. They most frequently arise during the relapse, but may be delayed till the period of convalescence. Usually they commence suddenly and may be preceded by rigors. In some cases there is also severe abdominal pain and vomiting. In some epidemics diarrhœa has been a frequent cause of death.

Pregnant women when attacked by relapsing fever almost invariably miscarry. The *abortion* may occur during the primary attack or the relapse. In advanced pregnancy the child is still-born or lives only a few hours.

The most common complications of the convalescent stage are pains in the muscles and joints, and ophthalmia.

The *pains in the muscles and joints* may be present during both the febrile periods, but are more often met with in the remission and after the relapse. They shift about from place to place, are often very severe, and the affected part is tender on pressure. There is rarely articular swelling or redness.

Ophthalmia is rather a sequel than a complication, for its onset is not usually observed for several days or months after the attack. The first symptom is dimness of vision, which is quickly succeeded by signs of inflammation in the iris and sclerotic. According to the observations of Estlander, the inflammation commences in the choroid and ciliary body, and, extending to the vitreous, gives rise to the dimness of vision (amaurotic stage). Later the other ocular structures are involved (inflammatory stage). This complication is very common. It is met with especially in those who are not properly fed and cared for during the period of convalescence. It occurs at all ages, but is most common between ten and thirty. Except in rare cases one eye only is affected, the right most frequently. Recovery is very slow, and sometimes permanent blindness results.

Anæmia is also a common sequel of relapsing fever. During convalescence purulent *otorrhæa* may arise. *Paralysis* of one or more limbs has occasionally been observed.

Protection.—Only a limited protection is afforded, for second attacks are not uncommon.

Morbid Anatomy.—The spleen is much enlarged and softened, and often contains infarcts or multiple small abscesses. The liver is also enlarged, and its cells are swollen and granular. Sometimes necrotic and inflammatory patches can be seen scattered through the

organ. Similar patches are found in the red marrow of the bones. The heart muscle undergoes fatty degeneration, especially if death has occurred from collapse. Fatty degeneration is also, at times, found in the voluntary muscles, kidneys, and other organs. The lymph follicles of the intestine are swollen.

The *spirillum* of relapsing fever, *Spirochæta Obermeieri* (σπείρα, a coil; χαίτη, hair) is a micro-organism belonging to the class of Protozoa. It is a fine, spiral, flagellated filament, pointed at the ends, and 20 to 40 μ in length. It is motile, and, when examined in the living state, can be seen rotating upon its long axis, at the same time alternately lengthening and shortening. Its presence can be demonstrated by examining either the fresh blood directly under the microscope, or stained preparations. If the latter method is adopted the blood is fixed on a cover-glass in the usual way (see Chapter II.). The cover-glass is then placed in a 1 per cent. solution of acetic acid until the hæmaglobin is removed. After getting rid of the excess of acid by washing in water, the cover-glass is stained in aniline-water gentian-violet solution, again washed in water, dried, and mounted in Canada balsam.

The spirillum is found in enormous numbers in the blood during the primary attack and the relapse, appearing just before the temperature rises and disappearing just before the crisis. Generally the spirilla occur singly, but they may be massed together in little clumps. During the remission and after the second crisis they can no longer be found in the blood; and it is probable, from experiments on apes, that they collect in the spleen during these periods. In fatal cases they are absent from the blood after death, but can be seen in the necrotic areas

in the spleen, often enclosed in phagocytes where they undergo degeneration.

The spirillum has not yet been cultivated in artificial media.

Pathology. — The cause of relapsing fever is the spirillum just described. Not only can the organism be demonstrated in the blood in all cases of relapsing fever, but this disease can be produced in apes and in the human subject by the injection of blood containing spirilla. According to Heydenreich, the spirilla are most active in their movements at a temperature of 60° to 70° Fahr., and they die at febrile temperatures. Hence he has put forward a theory that the organism is killed or rendered harmless by the pyrexia of which it has been the cause. It is possible that spores, formed by the spirilla during the primary attack, accumulate in the spleen and the marrow of the bones, and that in those structures they germinate and produce a generation of organisms which, escaping into the blood, give rise to the relapse. It has not yet been ascertained how the spirillum is transmitted from the sick to the healthy; for its presence has not been demonstrated in any of the secretions or excretions, nor has it been found outside the body.

Diagnosis.—In cases of doubt the blood should be examined for the characteristic spirillum, the presence of which is diagnostic.

The diseases in this country for which relapsing fever is most likely to be mistaken are typhus, enteric fever, and small-pox.

In *typhus* the symptoms at the onset are not so severe, the initial rigors are not so marked, and during the first two days of the disease the pulse is less frequent and

the temperature less raised than in relapsing fever. In typhus fever the liver and spleen are not enlarged, jaundice is rare, and a characteristic rash is present. In relapsing fever a rash is exceptional.

The onset of *enteric fever* is nearly always gradual, and even when sudden the symptoms are never of the severity which characterises the commencement of relapsing fever. The rose-spots and the persistence of the pyrexia beyond the seventh day also serve as points of distinction.

The initial symptoms of *small-pox* may be severe. In both this disease and relapsing fever rigors and lumbar pain may be marked; so that if the two diseases were prevalent at the same time it would be difficult, without a microscopical examination of the blood, to make a diagnosis before the appearance of the characteristic eruption of small-pox on the third day.

Prognosis.—Compared with typhus and enteric fever relapsing fever is not a fatal disease. In Great Britain the average case-mortality has been 4 per cent, but in other countries the rate has been higher. In the Bombay epidemic of 1877 to 1879 it was 18 per cent.

The age of the patient is of importance. In early life a fatal termination is rare; and of 1,366 patients under thirty admitted to the London Fever Hospital only seven died, a case-mortality of 0·5 per cent. Above thirty the fatality increased gradually until it reached 12·5 per cent in patients over sixty.

The following occurrences are most serious: hæmorrhages into the skin or from the mucous membranes; suppression of urine; cerebral symptoms (coma, delirium, and convulsions); severe diarrhœa; and collapse. The two last-mentioned complications may occur during the

remission, and in cases that during the febrile period appeared to be mild.

Treatment.—Patients suffering from relapsing fever should be isolated in wards set apart for the purpose. No remedy has hitherto been discovered which will cut short the primary attack or prevent the occurrence of the relapse. Consequently attention must be directed to the treatment of symptoms as they arise. Pyrexia should be dealt with by one or other of the methods described in Chapter I. The bowels should be kept open by mild aperients, but active purging is to be avoided. With a view to promote a free action of the kidneys and thus prevent the occurrence of uræmia, Murchison advised the administration of nitrate or acetate of potash, dilute nitric acid, and tincture of digitalis. Should suppression of urine supervene, dry-cupping and mustard poultices to the loins should be employed, and the action of the skin promoted by hot-air baths or hot wet packs. Headache, sleeplessness, and delirium are relieved by cold effusions or by opium. For pain in the hepatic and splenic regions, warm fomentations should be applied. When collapse occurs stimulants should be freely administered. This complication is to be especially looked for about the time of the crisis, and in elderly patients. Failure of the heart's action, apart from any sudden collapse, likewise calls for the use of stimulants.

The diet should be such as is given in typhus fever. During the remission and after the relapse the appetite quickly returns.

The severe muscular and articular pains of convalescence are best relieved by external applications containing opium, combined with the internal administration of quinine, iron, and opium. For diarrhoea the various vegetable astringents,

or ipecacuanha and opium, may be given. Those patients who become affected with ophthalmia should be placed upon a nutritious diet. In the early stages leeches should be applied to the temples and atropine dropped into the eyes. Small doses of calomel, quinine, and opium should be given every four or six hours. Later, counter-irritation to the temples should be employed, and the patient put upon a mixture containing iron and quinine. When the spleen remains enlarged after the febrile period the patient should be kept in bed. Murchison states that the best remedies for this condition are sulphate of iron and quinine internally, and iodine or red iodide of mercury externally.

CHAPTER XVIII.

ENTERIC FEVER. TYPHOID FEVER.

ENTERIC FEVER is a specific disease, of which the chief symptom is continuous pyrexia of some weeks' duration, accompanied by prostration, usually by an eruption of rose spots, and often by diarrhœa. In fatal cases inflammation and ulceration of the lymphatic follicles in the intestines are found.

Etiology.—It is only within the last fifty years that the distinction between enteric and typhus fever has been established. During the last twenty years the number of cases in England and Wales, as judged by the mortality returns, has been gradually diminishing. This is no doubt due to the improvements in sanitation which have been introduced. The average annual death-rate from enteric fever per million living during 1871—1875 was 374; during 1876—1880, 277; during 1881—1885, 216; and during 1886—1890, 179.

Geographical Distribution.—The disease is endemic in all countries, and is perhaps more prevalent in temperate than in tropical climates. Although endemic, local epidemics are by no means uncommon.

Season.—In England it is much more prevalent during the autumn than at other times of the year, and the largest number of cases occur during the months of October and November.

Dissemination.—The infection is conveyed in quite a different manner from that of scarlet fever or small-pox ; for the virus is not given off by the breath, but is contained in the alvine discharges. Consequently the mode of infection is generally an indirect one, and the disease does not spread from patient to patient, as in other infectious diseases. In most general hospitals cases of enteric fever are treated in the general wards, and the disease does not spread if proper care is exercised.

The virus retains its vitality for a long time outside the body, and appears to be capable of multiplying in the soil, in sewage matter, in drinking water, and in milk. If we admit this, then the whole question of the *de novo* origin of enteric fever loses much of its importance. The chief argument in favour of such an origin is the fact that sporadic cases not infrequently arise in isolated houses, where no source of infection from a previous case can be ascertained. Negative evidence of this nature must always be received with caution, as a proof of the *de novo* origin of any infectious disease. A similar argument might be applied to scarlet fever or small-pox ; for in these diseases it is often impossible to trace the source of infection. It must be remembered, in this connection, that mild cases of enteric fever may be unrecognised, and yet convey infection.

Considering that the virus is contained in the evacuations, it is not surprising that epidemics can frequently be traced to *defective drainage*. Whenever the drinking-water is contaminated with sewage, or there is an escape of sewer-gas into the dwellings, there is always danger of an epidemic of enteric fever arising. The presence in the drains of the specific virus from a case of enteric fever, is a necessary factor in the development of an epidemic. People may drink water contaminated with sewage, or

inhale sewer-gas for years, without contracting the disease, so long as the sewage is not specifically contaminated ; but as soon as it becomes infected with the evacuations of a case of enteric fever an epidemic is almost sure to arise.

Extensive epidemics have frequently been traced to the *water supply*, and in most cases the water has been found, on chemical analysis, to be contaminated with sewage matter. But it must be admitted that drinking water may be infected with the specific virus, although to a chemical analysis it may appear to be pure. For instance, a celebrated epidemic at Caterham was traced to the contamination of a deep well with the dejecta of a workman suffering from enteric fever. The amount of faecal matter in the water could not have been sufficient to allow of detection by a chemical analysis. Possibly a bacteriological examination would have revealed the presence of the typhoid bacillus or the bacillus coli.

Apart from extensive epidemics, smaller epidemics have been investigated in rural districts, where the disease has ceased when a fresh water supply has been obtained. But the original water supply in the cases in question was derived from several different wells ; and it is not probable that each well was separately infected. The only explanation that can be given is, that the virus has entered and multiplied in the soil, and that from the latter the wells have been infected.

From observations that have been made in Munich, Buda-Pesth, and Berlin, there is reason to believe that in some localities the prevalence of enteric fever depends upon variation in the *level of the ground water*. This is no doubt due to an infection of the soil with the typhoid virus ; and in Munich a decrease of enteric fever has ensued upon the adoption of measures precluding the infection of the soil with

fæcal matter. The exact way in which variation in the level of the ground water acts in determining a prevalence of the disease is still a debatable question. We adopt the view that it acts by contaminating surface wells, the water of which is used for domestic purposes. In Buda-Pesth enteric fever is more prevalent when the ground water is rising, while in Berlin and Munich the reverse is the case. It is probable that local conditions determine whether a rise or fall is the principal factor in causing a pollution of the wells.

Several epidemics have been traced to the *milk supply*. In some outbreaks a direct source of contagion has been discovered in the fact that cases of enteric fever were existent at the dairy. In other outbreaks the water used in the dairy was contaminated by sewage matter.

Epidemics have also been traced to the contamination of other articles of diet, such as *lemonade, ice-cream*, etc.

Some evidence has lately been brought forward to show that the ingestion of uncooked *oysters* may give rise to an attack of enteric fever. It is supposed that the oysters become contaminated with the enteric virus in oyster-beds that have been laid down too close to the outfall of sewage into estuaries and similar places on the sea-coast.

The escape of *sewer-gas* into the dwelling rooms has, in some instances, been the only explanation of several cases of enteric fever occurring in the same house.

Direct infection.—It has already been stated that enteric fever differs from other infectious diseases in not spreading directly from individual to individual. There is thus but little danger in visiting patients suffering from the disease. Nevertheless, nurses in charge of enteric patients are not infrequently attacked. This is often due to the lack of proper care and cleanliness. The virus is introduced into the mouth by fingers soiled with fæcal matter, or sometimes

possibly by the inhalation of fæcal matter that has been allowed to dry on the linen.

Mode of entrance of virus.—From what has already been said about the conveyance of the disease, it follows that the usual mode of entrance of the virus is through the digestive tract. It would also appear that the entrance is sometimes through the respiratory tract, as in the cases where an escape of sewer-gas into the house has been the source of infection. But even in these cases it is quite possible that the sewer-gas has infected articles of food (milk, water, etc.), and that the virus has entered the body in the usual way by the alimentary canal.

Age and Sex.—The following Table shows the age, sex, and case-mortality of 9,223 patients admitted into the Metropolitan Asylums Board Hospitals during the years 1871 to 1894:—

<i>Ages.</i>	MALES.			FEMALES.			TOTAL.		
	Cases admitted.	Deaths.	Mortality per cent.	Cases admitted.	Deaths.	Mortality per cent.	Cases admitted.	Deaths.	Mortality per cent.
Under 5 .	156	19	12·2	131	18	13·7	287	37	12·9
5 to 10 .	612	56	9·2	597	51	8·5	1209	107	8·9
10 „ 15 .	1148	117	10·2	982	164	16·7	2130	281	13·2
15 „ 20 .	1081	158	14·6	983	207	21·1	2064	365	17·7
20 „ 25 .	707	152	21·5	681	127	18·6	1388	279	20·1
25 „ 30 .	507	122	24·1	413	87	21·1	920	209	22·7
30 „ 35 .	296	87	29·4	258	50	19·4	554	137	24·7
35 „ 40 .	159	48	30·2	162	41	25·3	321	89	27·7
40 „ 45 .	90	25	36·0	85	19	24·2	175	44	30·0
45 „ 50 .	46	22		58	14		104	36	
50 „ 55 .	21	9		22	5		43	14	
55 „ 60 .	7	3	30·0	8	4	30·0	15	7	30·0
and upwards	8	3		5	1		13	4	
Totals .	4838	821	17·0	4385	788	18·0	9223	1609	17·4

From this Table it will be seen that the disease is not common in children under five years of age ; that it is most common between the ages of five and twenty-five ; that after the age of twenty-five the numbers gradually diminish ; and that persons over fifty years of age are rarely attacked. Males are slightly more liable to the disease than females. No age is exempt ; and cases are recorded in children under one year. The same Table shows that the total fatality is lowest between five and ten years of age ; that it is higher under five years ; and that it increases after ten years with each quinquennial period. Between the ages of ten and twenty the fatality is higher among females than males ; but in all later age-periods the reverse is true.

Incubation.—It is difficult to determine the length of the incubation period. It is probably about a fortnight, but it may be shorter ; and in a well-authenticated case it appeared to be only five days. 14
5

Clinical History. *Onset.*—The disease almost invariably begins insidiously, and it is usually some days after the onset that the patient first seeks medical advice. For this reason it is rarely possible to ascertain the exact day when the attack commenced ; but sometimes the onset is marked by a sudden attack of vomiting or headache, or even by a rigor.

Early symptoms.—Headache is generally one of the most pronounced of the early symptoms. In addition there is malaise, anorexia, nausea, and pyrexia. Epistaxis, slight hæmorrhage from the pharynx, and soreness of the throat are not uncommon, and cough is often a prominent symptom. It is unusual for the bowels to act normally, and there is generally either diarrhœa or constipation.

Later symptoms.—As the disease progresses the symptoms

become more characteristic, and about the end of the first week the rash appears.

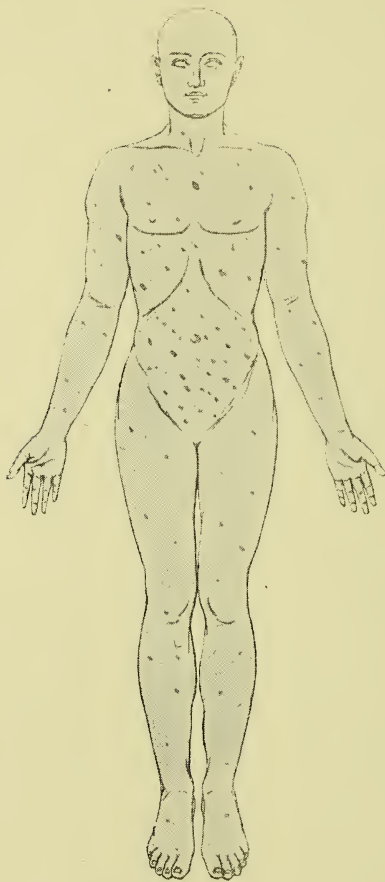


DIAGRAM XIV.—ENTERIC FEVER.

An eruption of rose-red, slightly raised spots; most frequent upon, and often limited to, the abdomen and chest. Occasionally seen sparsely upon the limbs; not often upon the face.

The rash is seen at first upon the abdomen, the lower part of the chest, and the back, and is often limited to

these parts. Sometimes it spreads all over the trunk and extremities, and in rare cases the face is also affected.

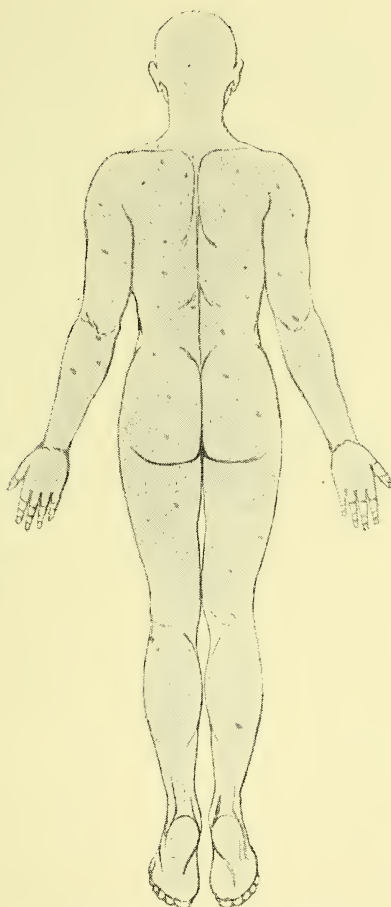


DIAGRAM XV.—ENTERIC FEVER.

It consists of small pink lenticular papules, about two lines in diameter, which fade on pressure. They are slightly raised above the surface, and can be felt by

running the finger lightly over the skin. It is often difficult to distinguish them from ordinary pimples. The

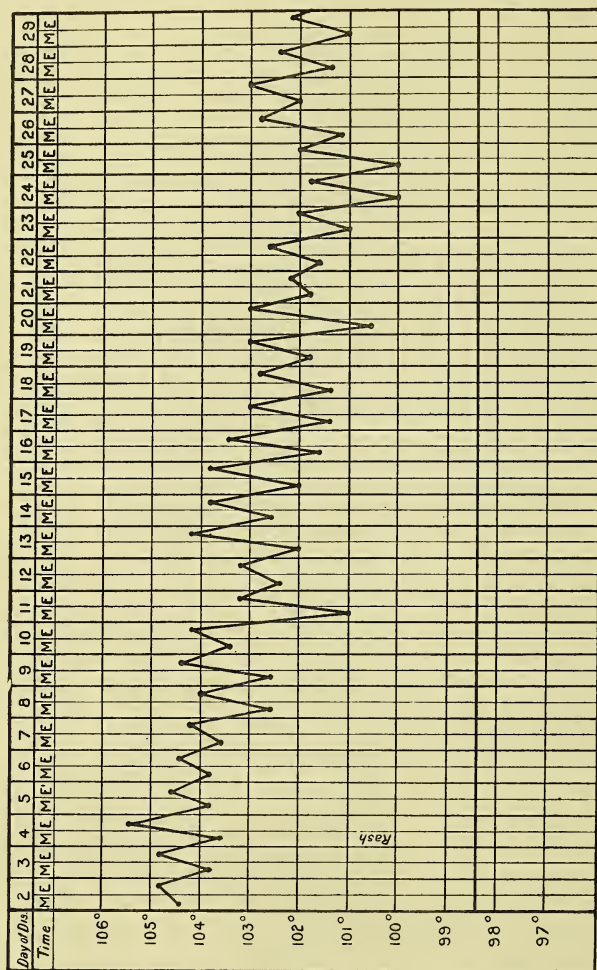


CHART U.

Female, aged 26. Prolonged attack of enteric fever; symptoms, however, never severe. There was mostly constipation all through.

papules are usually discrete and sparsely distributed, and number, as a rule, about a dozen or twenty; but several hundreds may be present. They always come out in

successive crops, each crop lasting a few days, and then fading. In some rare cases the spots are much larger than has been described. When the temperature falls no

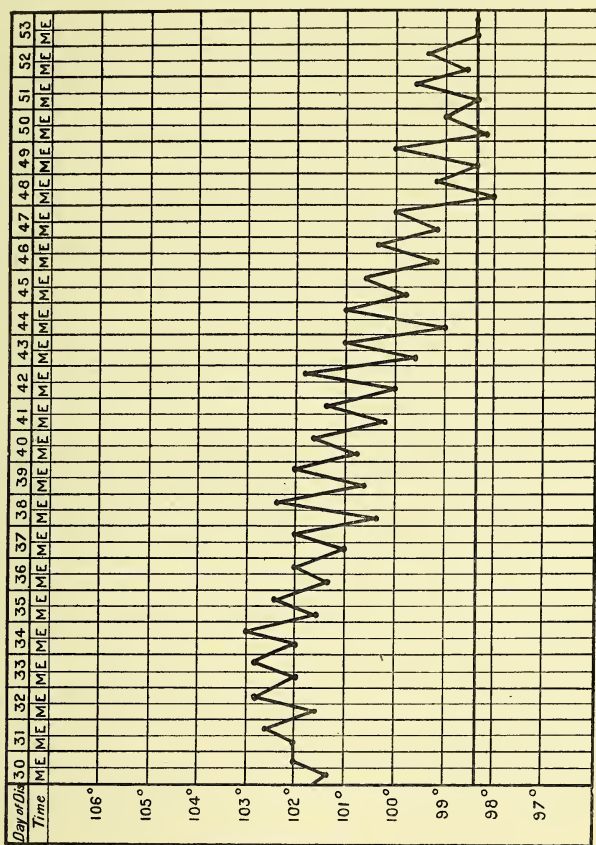


CHART U. (continued.)

fresh spots, as a rule, appear; but they may continue to come out for some days longer. In not a few cases the rash is entirely absent. Sudamina are frequently present. Sometimes the characteristic rash is preceded by a faint diffuse erythema over the chest and abdomen.

The course of the *pyrexia* may be divided into three stages—the initial stage, during which the temperature rises to its maximum; the fastigium, during which

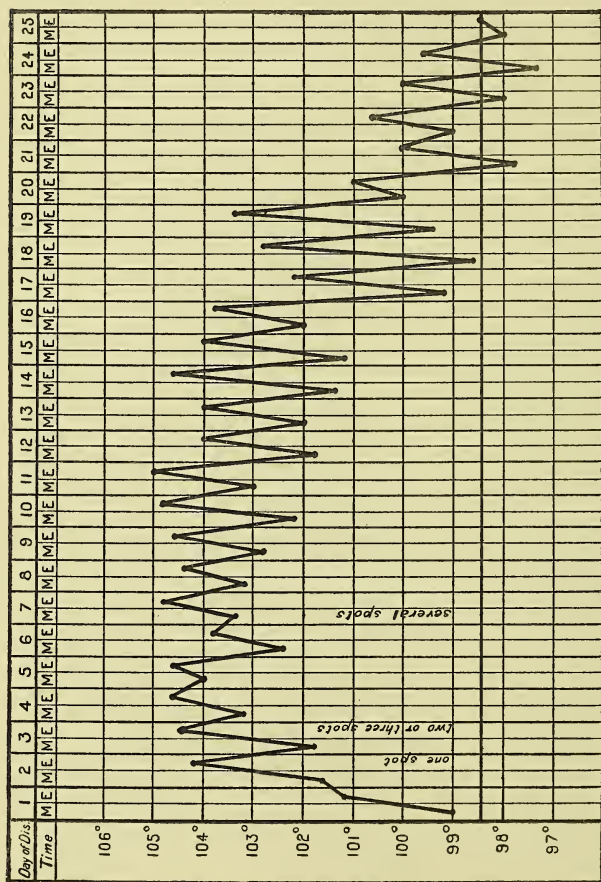


CHART V.

Female, aged 19. Very severe, but uncomplicated, case of enteric fever. Spots were present till the twenty-sixth day of the disease. Diarrhoea after the ninth day.

it remains near the maximum; and the stage of defer-
vescence.

The initial stage is usually described as possessing characteristic features. The temperature is said to rise in

a zigzag fashion, with morning remissions of about one degree, and evening exacerbations of two degrees, the result of which is that the maximum is reached in three or four days. But in cases where we have had the opportunity of watching the temperature from the beginning of the disease it has risen much more rapidly, and the maximum has been reached in one or two days. The zigzag rise is sometimes well marked at the commencement of a relapse.

During the fastigium the temperature remains between 102° and 105° Fahr., according to the severity of the attack, and there are morning remissions and evening exacerbations of one or two degrees. The length of the fastigium is very variable. In cases of moderate severity it lasts fourteen to twenty-one days, but in severe cases it is often longer, and in mild cases shorter.

The character of the temperature in the stage of defervescence is almost always the same. At first the morning temperature falls every day two or three degrees, but the evening temperature continues to rise to the maximum. Then the evening temperature shows a remission, and after some four or five days it reaches the normal point. For several days afterwards the temperature is often subnormal.

The tongue is at first covered with a white fur, except

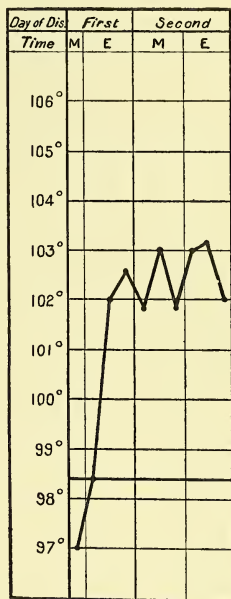


CHART W.

Showing sudden rise of temperature at the beginning of an attack of enteric fever. Temperature normal at 2 p.m.; 102° at 6 p.m.

at the tip and edges, which are red. At a later period the fur clears off, and leaves the tongue red, dry, and glazed. In severe cases it becomes covered with a dry brown fur, and is often cracked and furrowed.

There may be constipation throughout the illness, or attacks of diarrhœa and constipation may alternate. Diarrhœa is frequently severe, and there may be as many as a dozen evacuations during the day. When there is diarrhœa the appearance of the motions is characteristic. They are yellow and fluid, and resemble pea-soup in colour and consistence. In a few cases the stools are green. During the later stages of the disease yellowish sloughs may sometimes, though rarely, be detected. The presence of blood in the motions will be considered under the heading of Complications.

The abdomen is full and tumid, and is often tender and painful. In severe cases it becomes tympanitic, from the distention of the intestines with gas. Pronounced tympanites should give rise to the suspicion of peritonitis. A gurgling sensation can often be felt on pressure over the cœcum, but is of no diagnostic importance. The spleen, in the majority of cases, is enlarged, and can be felt on palpation. The fingers are placed in the left hypochondrium just below the ribs, the patient is told to take a deep breath, and the enlarged spleen is felt as it descends. It is generally very soft, and may thus escape detection. Percussion will then sometimes reveal the enlargement; but much reliance cannot be placed upon this method. In certain cases the spleen feels so hard as to suggest some chronic enlargement; but in these cases, as in the cases where it is soft, the enlargement disappears as the temperature falls.

The usual mental condition is one of lethargy and

drowsiness; the patient is listless and apathetic, and takes but little heed of what is going on around him. A quiet muttering delirium is often present, but there may be active delirium, so that the patient requires to be restrained. In severe cases coma may occur. Headache during the first few days is a prominent symptom, but it entirely disappears as the disease progresses. The pupils are generally dilated. Deafness in one or both ears is commonly present, but it passes off during convalescence. The throat is often sore, and the fauces reddened and covered with sticky mucus.

Muscular prostration is a marked feature of the disease. If the attack is at all severe the patient can hardly turn in bed, and tremor of the muscles occurs during attempts at movement. Subsultus tendinum is frequently present. The muscles often become wasted to an extraordinary degree, and a peculiar form of degeneration has been described. In addition to the muscles other tissues undergo wasting, and the subcutaneous fat rapidly disappears.

The pulse is rapid and compressible, and is much affected by any slight exertion. In severe cases it is dicrotic, and the first sound of the heart becomes short and feeble.

Bronchitis is almost invariably present, and is shown by the presence of râles and rhonchi, especially at the bases of the lungs.

The urine presents the usual character of the febrile state, being high coloured, acid, and depositing urates. A trace of albumen may be present. It often gives Ehrlich's reaction. This test is applied in the following way: Twenty-five parts of a saturated solution of sulphanilic acid in dilute hydrochloric acid (1 : 20) are added to one part of a 0.5 per cent. solution of sodium nitrite.

To this mixture is added an equal bulk of urine, and it is rendered strongly alkaline with ammonia. A ruby-red colour is produced. The test is not diagnostic, for the reaction may be given with the urine of other diseases, and is not always found in enteric fever. Retention of urine is not uncommon, and may require the use of the catheter.

Convalescence is exceedingly slow. It may be months before the patient has regained weight and strength. Some patients never regain their former vigour, and this is especially the case in older patients. Constipation often gives rise to much discomfort. During convalescence a number of sequelæ may arise.

TYPES OF THE DISEASE.—The course taken by enteric fever is exceedingly variable. We shall give a short account of several types.

Cases of moderate severity.—During the first few days the principal symptoms are malaise, headache, anorexia, a furred tongue red at the tip and edges, and perhaps diarrhœa. Towards the end of the first week the rash appears, and by this time the diagnosis can generally be made. The symptoms now increase in severity. The patient becomes dull and apathetic, the temperature keeps at about 103° , with morning remissions, and the pulse becomes soft and compressible. There is often moderate diarrhœa. There may be delirium, and there is always prostration and wasting. The rash continues to come out in successive crops. This condition continues until towards the end of the third week, when the temperature begins to fall, and becomes normal in a few days. With the fall of temperature the other symptoms subside, the diarrhœa stops, and spots no longer appear.

Severe cases.—The severity of the attack does not

generally show itself until the end of the first or the beginning of the second week. The pulse becomes rapid—130 to 160 per minute—and is compressible and dicrotic. The temperature keeps high (104° to 106°), and the morning remissions are slight. Diarrhœa is frequently profuse. The tongue is dry and cracked, and the teeth and lips are covered with sordes. There is extreme muscular weakness, subsultus tendinum is marked, and the patient soon falls into the "typhoid state." In this condition he lies helplessly upon his back, and takes no notice of his surroundings. When liquid food is placed in his mouth he swallows, but there is danger of the food getting into the larynx. From this state he may pass into a condition of coma, or may die from asthenia. Death commonly occurs during the third or fourth week, but not infrequently later. It may take place as early as the second week. When recovery ensues the temperature begins to fall about the end of the fourth week, but sometimes earlier. Convalescence is always protracted, complications and sequelæ are frequent.

Mild cases.—Enteric fever may be so mild that it is not recognised until some complication arises. Patients have been known to be seized with symptoms of perforation while at their ordinary occupation, having previously only felt a little out of sorts. Again, it is not uncommon for patients to seek admission into hospital just as the disease is terminating. They have been ailing for some weeks with malaise, but have not been sufficiently ill to seek medical advice. The temperature falls shortly after admission, and the occurrence of a relapse is perhaps the only evidence of the nature of the illness. In other cases the usual symptoms—spots,

diarrhœa, and fever—are present, but are not severe. Before the end of a fortnight the temperature falls to normal, and the disease terminates.

Enteric fever in children.—In children the attack is generally mild, and the symptoms are not so characteristic as in adults. Many cases terminate by recovery before the end of the second week. The temperature curve often shows marked morning remissions throughout the attack, and, corresponding to this, the general condition of the patient is much worse at night than in the day-time. Bronchitis is often severe, and the sputum may be streaked with blood. Emaciation is a prominent symptom, and the aspect of the patient is like that of a case of phthisis. Hæmorrhage from the bowels and perforation are very rare complications.

Complications.—Enteric fever is remarkable for the frequency with which complications arise. They are more commonly met with in severe cases, but even the mildest cases are not exempt. A fatal termination is often caused by one of the complications.

Hæmorrhage from the bowel is the most common complication. In 40 (7·9 per cent.) of the 506 cases which were under treatment at the Eastern Fever Hospital during the years 1892-94, there was hæmorrhage of sufficient intensity to require treatment. The hæmorrhage may be slight, and only consist of a few clots of blood in the motions, or it may be so profuse as to be fatal. As a rule, a single hæmorrhage is not fatal; and when death occurs, it is generally caused by repeated losses of blood in a patient already enfeebled by the poison of the disease. When the hæmorrhage is at all profuse the blood is fluid; and it may be of a bright red, or of a dark colour. The source of the blood is usually the capillaries of the inflamed

follicles, but it is sometimes a large vessel that has become ulcerated. Slight hæmorrhages may occur at any time, but profuse hæmorrhages not before the end of the second week. The occurrence of a profuse hæmorrhage is indicated by a sudden fall of temperature, in addition to the usual symptoms of loss of blood—a rapid pulse, pallor, restlessness, and sighing respirations. All these symptoms may occur before the blood has actually been evacuated.

Peritonitis and Perforation.—Peritonitis may be due to the direct spread of inflammation from the lymphatic follicles, to a sloughing or suppurating mesenteric gland, to a sloughing infarct in the spleen, or to a suppurating gall bladder. The most common cause is a perforation of an intestinal

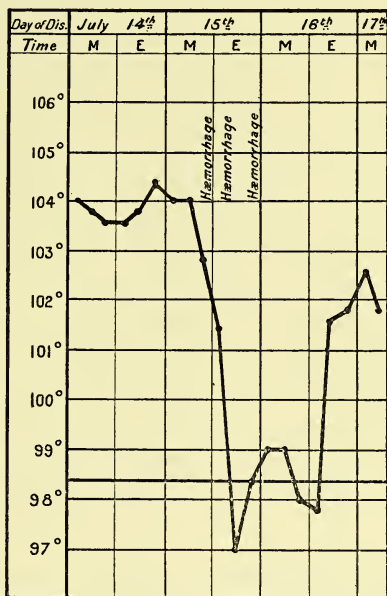


CHART X.

Chart showing effect of copious hæmorrhage on the temperature. On July 15th, at noon, about 9 ozs. of blood were passed; at 2 p.m., a pint; a little during afternoon; 10 ozs. at 10.30 p.m.; and a little at 2 a.m. on 16th.

ulcer, leading to the extravasation of fæcal matter into the peritoneal cavity. Perforation occurred in 16 (3.1 per cent.) of the 506 cases at the Eastern Hospital.

Whatever may be the cause of the peritonitis, the symptoms depend very much upon the general condition of the patient. If he is delirious or unconscious, even a perforation may occur without giving rise to any special

symptom, and may only be discovered at the post-mortem examination. If the general condition of the patient is good at the time of the onset of the peritonitis the symptoms are characteristic, and consist of abdominal pain, vomiting, and tympanites. The abdomen is tender, and does not move during respiration; and the patient lies on his back with the legs drawn up, with the face pinched, and eyes sunken. Recovery from such a condition is rare, but not impossible.

The symptoms of perforation may come on quite suddenly. There is severe, agonising pain, attended with great collapse; and cases have been recorded of death occurring within a few minutes. In the majority of cases the patient recovers from the initial collapse, the temperature, which had previously fallen to below normal, rises again, and symptoms of peritonitis set in. Sometimes characteristic symptoms are absent, and the insidious onset of peritonitis is the only indication of the occurrence of a perforation. The presence of gas in the peritoneal cavity, after perforation, may be sometimes detected by an abnormal resonance over the liver. The result of perforation is almost invariably death within two or three days; but one or two instances of recovery from the symptoms of perforation have been recorded. Sometimes the immediate cause of a perforation is straining at stool, or a violent movement of the body occurring during delirium. It is rare for a perforation to occur before the third week of the disease. Murchison lays stress on the liability of perforation to occur during convalescence.

Diarrhœa may be so profuse as to rank among the complications, although it rarely endangers life.

Bronchitis should be considered rather a symptom than

a complication ; and, in severe cases, there is always *hypostatic congestion* of the bases of the lungs. *Lobar* and *lobular pneumonia*, *pleurisy*, with or without effusion, and *empyema*, are not uncommon. *Pneumothorax*, from a breaking-down infarct or patch of lobular pneumonia, is a rare complication. *Ulceration of the larynx* is occasionally met with in fatal cases, but it does not often give rise to symptoms during life. The ulceration rarely causes a necrosis of the cartilages, or œdema of the glottis.

Phlebitis is not uncommon, and usually affects the veins of one of the lower extremities, and the leg becomes swollen and tender. Should the clot become detached, a pulmonary embolism is the result.

Parotitis, usually suppurative, is met with not infrequently.

Degeneration of muscles.—The muscles are always wasted, and a peculiar form of degeneration has been described. Recovery ultimately ensues ; but it may be months before the patient is able to walk properly.

Bed sores are liable to occur unless great care is taken.

Of the rarer complications we must mention *boils*, *abscesses* in various parts, *pyæmia*, *otitis media*, *optic neuritis*, *peripheral neuritis*, *localised gangrene*, *tubal* or *suppurative nephritis*, *diphtheria*, *hæmaturia*, and *purpura* with hæmorrhages from the various mucous membranes. Occasionally the *heart* becomes dilated. *Endo-* and *pericarditis* are rare.

Sequelæ.—*Periostitis* and *ostitis*—especially affecting the tibiæ and ribs—are not uncommon. Painful nodes form over the bones, and may either subside or suppurate ; in the latter case there may be necrosis of the bone. These conditions may first manifest themselves many months after the attack of enteric fever.

Various *mental affections* may follow. The most

common is a loss of memory, which usually passes off after a time. It may, however, be permanent, and sometimes is

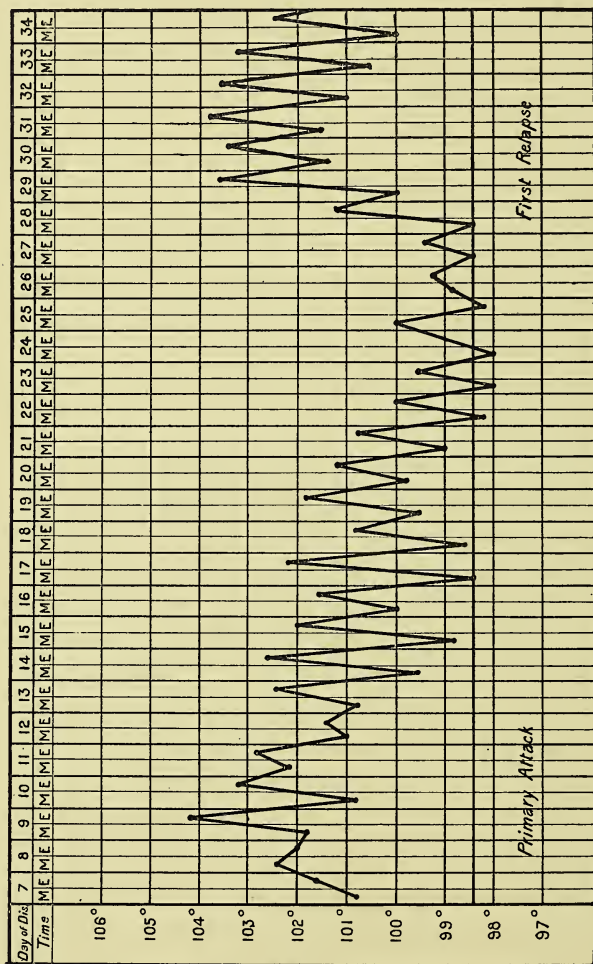


CHART Y. (continued on pages 311 and 312.)

Case of enteric fever, with two relapses, in an adult female.

associated with a condition of dementia. More rarely an attack of acute mania develops during convalescence.

Marasmus sometimes occurs, even when the patient

is taking food well, and the temperature is normal. Recovery usually follows, but it may end fatally.

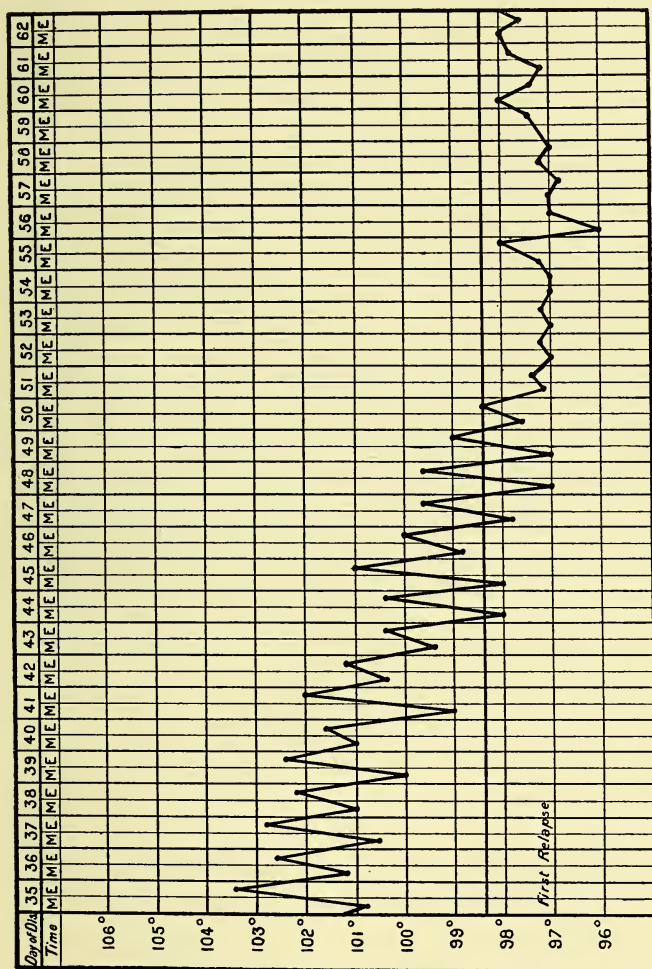


CHART Y, (continued.)

Sometimes it appears to be kept up by the administration of alcohol, and the patient recovers when this is discontinued.

Relapses are by no means uncommon. Of the 506 cases mentioned above, 66 suffered from relapse—an incidence of 13 per cent. The time at which a relapse occurs is usually within a fortnight after the temperature has become normal, but it may begin as late as the end of three weeks from this time. In some cases it

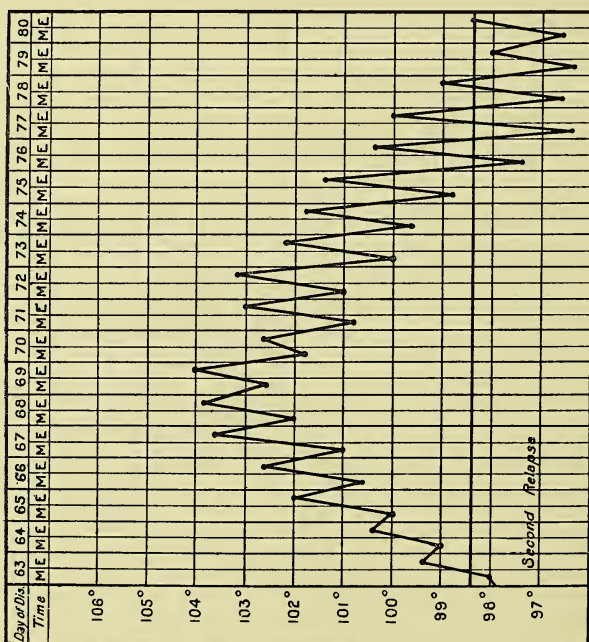


CHART Y. (continued.)

overlaps the primary attack. Towards the end of the third week of the disease the temperature begins to fall, as if indicating the commencement of convalescence, but before reaching the normal rises again, and the disease is prolonged for another fortnight or three weeks. Such cases are often looked upon as cases of prolonged enteric fever; but there is little doubt that they are relapses,

beginning before the primary attack has ended. A relapse runs through a similar course to a primary attack, and is attended by the same signs and symptoms. It is generally milder, and of shorter duration ; but it may be severe, and even end fatally. The relapse is accompanied by a fresh inflammation of the lymphatic follicles in the intestine. Two, three, and even four relapses may occur.

During convalescence it is not uncommon to meet with irregular and transient elevations of temperature, which must be distinguished from true relapses. Such rises may be due to septic absorption through the ulcerated bowel.

Protection.—An attack of enteric fever certainly confers very considerable protection ; but, nevertheless, second attacks have been known to occur.

Morbid Anatomy.—The most constant and characteristic lesions observed after death from enteric fever are inflammation and ulceration of the lymphoid follicles of the ileum. The appearances depend upon the stage of the disease.

In a case fatal during the height of the disease, in the *third or fourth week*, the lower two or three feet of the ileum present ulcers having the following features. They are situated in the Peyer's patches or the solitary follicles, and their edge is somewhat ragged and undermined, and is often surrounded by a zone of swollen, pink-coloured lymphoid tissue. The floor of the ulcer is occupied by an irregular bile-stained slough, more or less adherent. Where the slough has separated the floor of the ulcer is formed by the exposed muscular tissue of the intestine. Occasionally the muscular tissue is affected by the sloughing process, and the peritoneum is exposed. The ulceration is usually at a more advanced stage

near the ileo-cæcal valve; here the sloughs may be found entirely shed, while higher up they may be only partly, or not at all, separated. Though the ulcers are usually confined to the Peyer's patches and the solitary follicles, it is not uncommon to find them extending into the surrounding mucous membrane; and this is especially the case on and near the valve. In severe cases nearly the whole of the mucous membrane of this part of the intestine may be ulcerated. Sometimes two or three ulcers are found on one Peyer's patch, separated from one another by swollen and inflamed tissue. The number of ulcers varies greatly. Occasionally every Peyer's patch and solitary follicle in the last six or seven feet of the small intestine is affected, but sometimes only a single ulcer is found. Usually about six to ten ulcers are distributed through the last two or three feet of the ileum. The solitary follicles of the large intestine are usually ulcerated only when the small intestine is severely affected; and here, too, the ulcers are chiefly situated in the cæcum and ascending colon—that is, in the neighbourhood of the ileo-cæcal valve. It is very rare to find them in the sigmoid flexure or rectum, and only exceptionally is the large intestine alone affected. Still more rare is it to find, in cases fatal during the third or fourth week, no ulceration at all, either in the small or the large intestine. But such cases have been recorded. In cases where there has been hæmorrhage blood-clots can be found adherent to the bases of the ulcers, but it is uncommon to be able to find the exact source of the hæmorrhage when the latter has been the immediate cause of death.

The mesenteric glands are much swollen and inflamed, and present a pink, fleshy appearance on section. They

may be softened in the centre, and even purulent. The spleen is enlarged to two or three times its normal size, and is, as a rule, soft, and even diffuent. The liver and kidneys may be somewhat swollen, but to the naked eye show nothing else amiss. The lungs show marked hypostatic congestion of the bases.

If death has occurred at an earlier date the intestinal lesions are different. At the end of the *first week* the Peyer's patches and solitary follicles are intensely swollen and inflamed, and in those near the valve there may be detected signs of commencing necrosis.

During the *second week* the necrotic process has advanced still further, and sloughs are seen adherent to the underlying tissue.

If death has occurred after the *fourth* or *fifth week* the ulcers may be found in a state of repair, varying according to the period at which death has ensued. The slough separates piecemeal, rarely *en masse*, and leaves an ulcer with the floor formed by the muscular coat or the peritoneum, and with a well-defined and undermined edge. At this stage the inflammation has subsided, and the edge of the ulcer is little, if at all, raised. Later, minute granulations appear on the floor of the ulcer, and, at a period varying according to the nature of the case, the ulcer is completely filled up by new tissue. The whole of the tissue appears to be renewed; for, when death has occurred a few weeks or months after an attack of enteric fever, the most that can be seen is the "shaven-beard" appearance, the patch or follicle showing numerous minute black dots, and being usually of a dark-greyish colour. Little or no cicatricial tissue is formed; hence a constriction of the intestine never results.

When death has taken place during a *relapse* two stages are seen in the follicles. In some there is recent inflammation and sloughing, corresponding to the parts affected during the relapse, and in others there are ulcers from which the slough has just separated, or which are in a state of repair, corresponding to the parts affected by the primary attack. The two stages may even be seen in the same Peyer's patch.

In cases where the disease has run a *protracted course* (five to seven weeks), without a definite relapse, the two stages may still be seen, and the most advanced ulceration is in the neighbourhood of the ileo-cæcal valve.

Perforation usually occurs in an ulcer situated in the lower two feet of the ileum, but occasionally higher. In by far the majority of the cases the aperture is round and minute, and is caused by the extension of the sloughing process to the peritoneum. Sometimes a longitudinal slit is found, looking as if the thin base of the ulcer had been torn. It is a rare occurrence to meet with a large perforation. Peritonitis, when due to perforation, is exceedingly acute. A foul, brown-coloured, purulent fluid is found in the pelvic and lumbar regions. The peritoneum is covered with flakes of lymph, binding together the adjacent coils of the intestine, and the peritoneal vessels are dilated. The inflammation is most marked near the seat of the perforation, which may be sealed up with lymph. Peritonitis may be also due to the rupture of a suppurating mesenteric gland, or to a direct extension of the inflammation from the intestine without perforation.

Ulceration of the larynx is occasionally found. It generally affects the mucous membrane over the internal

surface of both arytenoid cartilages and the posterior portion of the vocal cords. The ulceration may extend deeply, and expose the cartilage. The kidneys are, in a few cases, the seat of an acute parenchymatous or a suppurative inflammation. The latter condition may occur quite apart from cystitis. Infarcts are sometimes found in the spleen. The voluntary and heart muscles undergo a form of degeneration. Rupture of the muscles of the limbs has been recorded.

Microscopical Anatomy. — Sections of the affected lymphoid follicles in the intestine and the mesenteric glands reveal the ordinary appearances of inflammation and its results. In the liver and kidney minute necrotic areas, and foci consisting of accumulations of leucocytes, are frequently found. The fibres of the voluntary muscles are often degenerated, the striæ disappearing, and the fibres becoming either granular or abnormally translucent and waxy. The muscular fibres of the heart are frequently granular.

The "Typhoid" Bacillus. — The Eberth - Gaffky or "typhoid" bacillus is present in the spleen, the mesenteric glands, and the inflamed lymphoid follicles of the intestine. It is sometimes found in the urine. Its presence can be readily demonstrated in sections of the spleen or mesenteric glands stained with carbolic methylene blue. The bacilli are found arranged in little clumps, scattered irregularly through the section. In the lymphoid follicles it is more difficult to distinguish the bacilli, on account of the presence of other bacteria. Cultivations can easily be obtained from the spleen and mesenteric glands, but with difficulty from the intestinal evacuations.

The bacillus is a short, thick, rod-shaped micro-organism, which sometimes becomes elongated, forming threads. It

stains well with carbolic methylene blue ; but is decolourised by Gram's method. Unstained portions, looking like spores, can sometimes be seen. These are not true spores ; they are only vacuoles, caused by contraction of the protoplasmic contents. The bacillus is motile, and special methods of staining reveal the presence of flagella, attached all over the surface.

The bacillus grows well on all the ordinary media, and at the usual temperature of the air. The appearance of the cultivations is not very characteristic ; and it is only by a careful study of its life-history that the bacillus can be distinguished from several other bacilli, notably the bacillus coli. On the surface of gelatine the growth assumes the form of a thin layer, with crenated edges. The gelatine is not liquefied. In plate-cultivations the superficial colonies in three or four days appear as translucent plaques with crenated edges, and under the low power of the microscope the surface is seen to be markedly striated. The growth on potato is often, though not always, characteristic. To the naked eye the surface of the potato appears moist, without apparent growth ; but a microscopical examination of the material taken from the surface shows that an abundant growth has taken place.

Both in morphology, and in manner of growth, the "typhoid" bacillus closely resembles the bacillus coli, a bacillus constantly present in the intestine of man and other animals. The principal points of distinction between these bacilli are the following :—The "typhoid" bacillus is rather longer than the bacillus coli ; it possesses greater motility, and is provided with more flagella. The bacillus coli curdles milk, the "typhoid" bacillus does not. The bacillus coli produces indol and bubbles of gas, the

PLATE III.

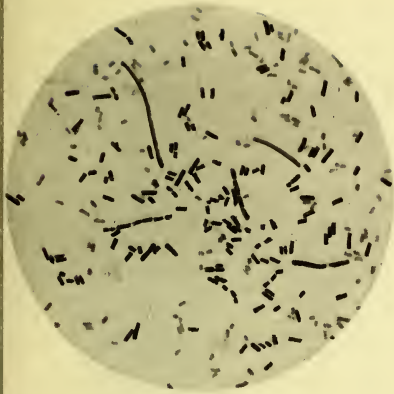


Fig. 1.—Typhoid bacillus.
Agar cultivation, 20 hours.
Gentian violet. $\times 1000$.

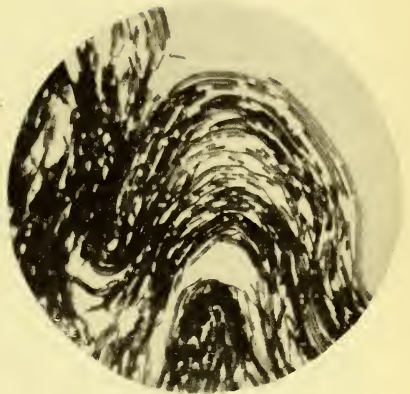


Fig. 2.—Typhoid bacillus.
Colony on gelatine, 72 hours.
Cover glass impression. $\times 500$.

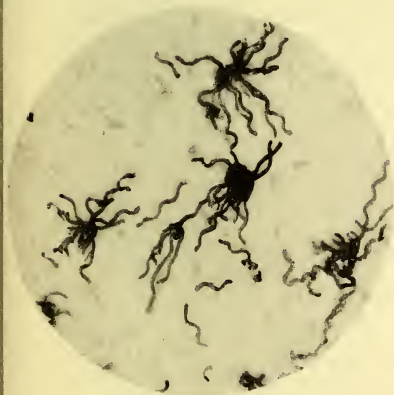


Fig 3.—Typhoid bacillus.
Flagella. Gentian violet. $\times 1500$.

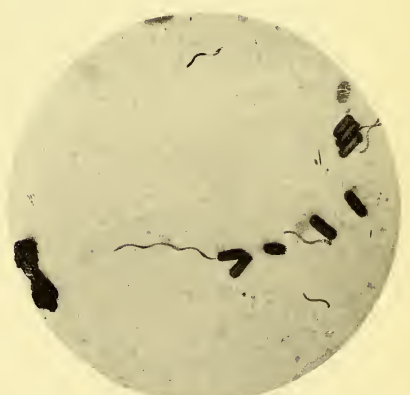


Fig. 4.—Bacillus Coli.
Flagella. Gentian violet. $\times 1500$.

"typhoid" bacillus produces neither indol nor gas. The bacillus coli grows more rapidly than the "typhoid" bacillus.

There are a number of varieties of the bacillus coli, differing from one another in mode of growth and microscopical appearances. Some of the varieties are exceedingly difficult to distinguish from the "typhoid" bacillus, while others can be readily distinguished by the points of difference above given. Both the bacillus coli and the "typhoid" bacillus, when injected in sufficient quantity into the peritoneal cavity of guinea-pigs, cause peritonitis, followed by death. The bacillus aquatilis sulcatus, which is not infrequently found in drinking water, may be mistaken for the "typhoid" bacillus, but it can be distinguished by its inability to grow at a temperature of 40° C.

Pathology.—The virus of enteric fever is undoubtedly a micro-organism. The mode of dissemination of the disease, its communicability by means of water or milk contaminated with the evacuations of infected patients, the occasional spread to those in attendance upon patients, and the protection afforded by one attack, point unmistakably to the nature of the virus.

There are good reasons for believing that the "typhoid" bacillus is the cause of the disease. The bacillus is constantly present in the spleen, mesenteric glands, and lymphoid follicles of the intestine of patients dying of enteric fever; and it has never been found in the tissues of the body in any other disease. There is also experimental evidence that similar effects to those of enteric fever can be produced in animals by the injection of living typhoid bacilli or their toxines. Death is caused in guinea-pigs by the subcutaneous inoculation of cultivations whose virulence has been exalted by repeated

passages through animals. The mucous membrane of the intestines after death is found inflamed, and the lymphatic follicles are swollen. Similar effects are produced by injecting the toxins freed from bacilli by filtration. Another interesting result of the inoculation is an enormous multiplication of the bacillus coli normally present in the intestine; and the meteorism that occurs in the human subject and in the experimental disease is doubtless due to the gases produced by this bacillus. The "typhoid" bacillus in the human subject grows in the lymphatic tissues of the intestine, and does not appear to multiply in the intestinal contents. If we accept the bacillus as the cause of the disease, we must consider the symptoms to be due to an absorption of the toxins.

As to the various secondary complications, such as periosteal abscesses, empyemata, etc., some are produced by the "typhoid" bacillus, and others by the secondary invasion of the pyogenic streptococci and staphylococci. When we consider the amount of ulceration of the intestines in a severe case of enteric fever, it is not a matter of surprise that a secondary invasion by pyogenic cocci frequently occurs. It is probable that such an invasion accounts for many of the symptoms, even in cases where there are no localised complications pointing to a secondary infection. The periosteal nodes and bone abscesses are interesting from the fact that they may arise long after convalescence has been established. The "typhoid" bacilli are almost invariably found in these foci, where they must have remained latent for a long period.

Some authors look upon the "typhoid" bacillus and the bacillus coli as simple varieties of the same species. This

view is strengthened by the fact that an animal which has been rendered immune towards the one bacillus is also immune towards the other. On the other hand, no one has yet succeeded in converting the one bacillus into the other. If we look upon the two bacilli as identical, we must consider that the bacillus coli, when it has invaded the tissues of the body, alters in character, and is converted into the "typhoid" bacillus. If this be the true view, then the real cause of enteric fever remains to be discovered.

The pathology of relapses can be explained in the same way as in other fevers (*vide* Chapter II.).

The "typhoid" bacillus has been found on several occasions in contaminated drinking water; it has usually been accompanied by the bacillus coli in much larger numbers. It must, however, be admitted that in many instances where the disease has spread through the water supply, a bacteriological examination has failed to reveal the presence of the "typhoid" bacillus. But in these cases the water has, from the large number of bacilli coli present, been proved to be contaminated by sewage matter.

Diagnosis.—During the first few days it is generally impossible to arrive at a diagnosis; and at first the case is often considered to be a bilious or indefinite febrile attack, or influenza. Headache, nausea, diarrhœa, and bronchitis, associated with pyrexia, should always arouse suspicion, especially if the attack has begun insidiously. A history of previous cases in the same house, or in the neighbourhood, is of importance. But in most cases it is necessary to wait until more characteristic symptoms have developed. When the spleen has become enlarged and the rash has appeared the diagnosis is easy. Unfortunately in many cases characteristic symptoms are completely

absent. There may be no diarrhœa, no enlargement of the spleen, and no rash, or only doubtful spots. Such cases are extremely puzzling, and the diagnosis must often be made by eliminating all other conditions which give rise to a long-continued pyrexia. Some fulness of the abdomen is generally present in enteric fever, and is of great assistance in forming a diagnosis.

The diseases mistaken for enteric fever are either general diseases or certain abdominal affections.

The abdominal affections comprise peritonitis from any cause, pelvic cellulitis, tuberculous peritonitis, perityphlitis, and ulcerative colitis. The distinction from *pelvic cellulitis* and *peritonitis* ought not to present much difficulty, except perhaps, in cases of enteric fever complicated with the latter affection. A careful inquiry into the history of the case will usually give sufficient indication of its nature. The same may be said about *tuberculous peritonitis* and *perityphlitis*. In the former disease ascites, enlarged glands, or matting of the intestines may be detected, and in the latter pain and induration in the region of the cœcum can be made out. *Ulcerative colitis* is sometimes difficult to distinguish, on account of the presence of diarrhœa, hæmorrhage from the bowel, and pyrexia. Pain is a prominent symptom, and the motions often contain pus and mucus. An examination of the rectum will sometimes reveal the presence of ulceration.

The general diseases which may be mistaken for enteric fever are typhus, influenza, pneumonia, meningitis, general tuberculosis, pyæmia and allied septic diseases, ulcerative endocarditis, phthisis, acute delirious mania, and, in some countries, malaria.

General tuberculosis is the disease which presents the greatest difficulties, especially as it is met with more

frequently in children, in whom the symptoms of enteric fever are often atypical. Continued pyrexia and wasting are common to both diseases, and in both the spleen may be enlarged. Rapidity of respiration and cyanosis should arouse the suspicion of tuberculosis, and search should be made for the presence of any local tuberculous mischief. The ophthalmoscope ought to be used, inasmuch as tubercles are sometimes to be found in the choroid.

Pyæmic conditions can generally be recognised by repeated rigors, or by the discovery of some focus of suppuration from which the disease has started. It should be remembered that there may be a deeply seated suppuration in connection with the bones or pelvic organs, or an inflammation of the ear or teeth, which can only be discovered on careful investigation. In *ulcerative endocarditis* there is usually evidence of cardiac disease, and the characters of the bruits may change while the patient is under observation. The spleen and other parts are often the seat of septic emboli, and the urine frequently contains blood and casts.

The delirium of *meningitis* may be mistaken for that of enteric fever. The severe pain, paralysis, retraction of the head, optic neuritis, and the fact that the pulse is at times slow, as compared with the temperature, will all assist in forming a diagnosis.

During its early stage enteric fever may present a great similarity to *influenza*, and it may be necessary to wait until the characteristic symptoms of the one or other disease have manifested themselves. The onset of influenza is more sudden, and its course shorter, than that of enteric fever.

Pneumonia is not difficult to distinguish from enteric fever, nevertheless a large proportion of the cases sent as

enteric fever to the Metropolitan Asylums Board Hospitals are cases of pneumonia. The sudden onset, the rusty sputum, the rapid breathing, the pain, and the physical signs, only make a wrong diagnosis justifiable when the patient is considered to be the subject of both diseases. In children enteric fever is sometimes mistaken for *phthisis*. The child is wasted, has hæmoptysis and cough, and there are, perhaps, doubtful signs at one apex. A careful examination will suffice to determine the diagnosis.

Acute delirious mania may be mistaken for enteric fever. There is pyrexia, emaciation, and delirium, combined with a dry, brown tongue, and with sordes on the lips. An inquiry into the history will often elicit the fact that for some time past the patient has shown signs of mental disturbance. The characteristic signs of enteric fever—spots and enlarged spleen—are absent.

Cases of enteric fever are sometimes mistaken for *diphtheria* or *tonsillitis*. The mistake is due to the fact that in enteric fever a complaint of sore throat is sometimes made, and, on inspection, the fauces are found reddened and covered with flakes of sticky mucus. *Acute gastro-enteritis* is sometimes mistaken for enteric fever, especially in children. Some forms of *malaria* assume the type of enteric fever, and in countries where both diseases are rife a mistake in diagnosis is frequently made. An examination of the blood for the malaria plasmodium will at once clear up the diagnosis.

The differential diagnosis between *typhus* and enteric fever has been discussed under the heading of the former disease (p. 274).

Prognosis.—From the Table on p. 294 it will be seen that the fatality varies between 8·9 and 30·0 per cent., according to the ages of the patients attacked. Broadly

speaking, it may be stated that the younger the patient the more chance he has of recovery.

A fatal termination may be due either to the severity of the attack or to the onset of some complication. The most common cause of death is by gradual cardiac failure. Consequently particular attention should be paid to the state of the pulse, and the character of the first sound of the heart. So long as the pulse is below 120 the case may be looked upon as a mild one ; but when the pulse reaches 130, and especially if it is weak and dicrotous, danger should be apprehended. A short and weak first sound of the heart must be looked upon as an unfavourable sign. In forming a prognosis from the state of the pulse the stage of the disease should be taken into account. A very frequent pulse during the first or second week is of much more significance than it would be towards the end of the third week, when the disease ought soon to show an abatement. Next in importance to the pulse is the temperature. If the temperature remains as high as 104° Fahr., with only slight morning remissions, the case should be considered to be severe. Again, if the temperature shows no signs of falling towards the end of the third week, the case is probably one in which a relapse will overlap the primary attack, and a continuous fever of some six weeks' duration is likely to occur. It will then be a question whether the patient's strength can last out such a protracted period of pyrexia. Under these circumstances, if the pulse is bad, the gravest prognosis should be given. Marked delirium, especially at an early stage of the disease, a presentiment of death, much prostration, the supervention of coma, the passing of the evacuations unconsciously, and subsultus tendinum, are all unfavourable symptoms.

The constitution of the patient is of importance in

forming a prognosis. Patients whose constitutions are undermined through the effects of alcohol are especially likely to succumb to the disease.

With regard to complications, the most serious are perforation and peritonitis. If there is distinct evidence of perforation the case is almost invariably fatal; nevertheless, one or two cases of recovery have been recorded. Peritonitis without perforation is a grave complication, but is not necessarily fatal. Slight hæmorrhage is not of much importance, but when it is pronounced anxiety must be felt; for although the hæmorrhage may not be sufficient to cause death from loss of blood, yet it is liable to recur, and thus to weaken the patient. Profuse diarrhœa of long duration is an unfavourable symptom, on account of its weakening effect, and because it indicates severe intestinal lesions.

The supervention of such complications as pneumonia, diphtheria, empyema, etc., is of bad import.

Treatment.—Save under special circumstances no patient suffering from enteric fever should, on account of the risk of perforation, be removed from his abode after the close of the first week of the disease. If he *is* to be sent to a hospital he should be removed early, even though there is a chance of the subsequent alteration of the diagnosis. In some cases the characteristic symptoms are not manifested till after the end of the first week; and if the practitioner waits for these symptoms before ordering the removal of the patient, he will also be waiting till that period of the disease when removal is most dangerous.

The patient should be kept in bed, in the recumbent position, until convalescence is well established. On account of the danger of perforation most careful nursing and dieting are necessary. The patient should not be

allowed to turn himself in bed, nor, on any account, to sit up. Bed sores must be prevented by cleanliness, hardening the skin with methylated spirit, dusting with zinc and starch powder, and by the use of a water bed to prevent pressure.

The diet should consist principally of milk, and should be given in small quantities every hour or two. For an adult, about three pints may be allowed in the twenty-four hours. It may be supplemented by beef tea or the various extracts of meat, custard, and jelly. To allay thirst, ice, soda water, lemonade made from fresh lemons, orange juice freed from pips, and imperial drink are useful. A little tea added to the milk will render it more palatable. If the milk is not digested—and this can be recognised by an examination of the stools—it should be peptonised, or lime-water added. Some patients absolutely refuse milk, and then recourse must be made to cocoa and various prepared foods. The diet during convalescence is also an important point, for there is danger of setting up perforation by injudicious feeding. The usual plan is to abstain from solid food until a week or a fortnight after the temperature has become normal, according to the severity of the case. Then cream, the crumb of bread, or carefully pounded meat mixed with gravy, may be given. In a few days a little fish, freed from bones, can be allowed, and at the end of the third week of convalescence a little chicken.

The temperature should be taken for at least a month after convalescence, and on any sign of a relapse the patient should be put back on a milk diet.

If the patient is seen early, before the end of the first week, it is well to give a couple of grains of calomel, to clear the bowel of solid contents. Except for this, drugs are only required as symptoms arise; for no drug

is known which will cut short the disease. When there is evidence of excessive putrefactive changes existing in the intestine, as shown by distention, intestinal antiseptics may be employed. Salol, in doses of ten grains every four hours, or β -naphthol, in doses of five to twenty grains, are useful intestinal antiseptics. When there is constipation in addition, a twelfth of a grain of calomel may be added to the salol. Care must be exercised in administering purgatives, for fear of causing perforation. Any strong purgative is decidedly contra-indicated. An accumulation of fæces in the intestines is injurious, and the bowels should be kept open by the use of enemata when necessary.

Stimulants should be given according to the state of the heart. As long as the pulse is fairly strong no stimulant is required ; but when it becomes weak and dicrotic alcohol should be given. As far as the amount is concerned, the previous habits of the patient should be taken into account ; and it may perhaps be advisable to give a little alcohol all through the illness to patients who have been previously in the habit of taking it. When no benefit follows the use of alcohol it should be discontinued. Sometimes, when the heart is failing, strychnine, ammonia, digitalis, or twenty-minim doses of spirits of camphor, given in port wine, are useful.

Delirium may be treated with a wet pack or with the continuous bath.

Slight hæmorrhage does not require treatment ; but should it be severe, measures must be taken to arrest it. The application of ice to the abdomen or the rectum is sometimes efficacious. The B.P. starch and opium enema, the administration of tannic or gallic acid combined with opium or turpentine, and the subcutaneous injection of ergotine, are all valuable.

Peritonitis should be treated by keeping the patient under opium, either given by the mouth or subcutaneously. The application of ice to the abdomen often relieves pain. When perforation occurs the patient should be fed entirely on nutrient enemata and suppositories, and morphia should be injected subcutaneously. This complication is almost invariably fatal under ordinary treatment; and the question must arise whether or not an attempt should be made to deal surgically with the aperture in the intestine. Such attempts have been made, but at present without success. If the perforation should occur in a patient whose condition was previously fairly good, an operation is certainly justifiable. Probably the best treatment would be to bring the perforation to the surface of the abdomen, and if the patient survives to deal with the artificial anus later.

Diarrhœa does not, as a rule, require active treatment. Unless it appears to be really exhausting the patient no attempt should be made to check it. When deemed necessary, the starch and opium enema, or opium given by the mouth, is generally sufficient.

In cases where the tongue is very dry and furred, and the mouth full of sticky mucus, the following mouth-wash will be found useful :—

Glycerine of borax	ʒi.
Tincture of myrrh	℥x.
Water to one ounce.					

For the treatment of pyrexia see Chapter I.

CHAPTER XIX.

ERYSIPELAS.

ERYSIPELAS is a spreading inflammation of the skin due to infection with the streptococcus erysipelatis. It is attended by pyrexia and other constitutional disturbances, and is contagious.

Etiology.—When looked at from a clinical point of view, erysipelas appears at first sight to be a definite specific disease with characteristic symptoms. Nevertheless there are many facts in connection with its etiology and pathology which show that it cannot be considered on quite the same footing as the other specific fevers. The latter diseases are specific in the sense that they “breed true.” A case of rubeola, for example, will in other persons give rise only to rubeola, and not to any other disease. A case of erysipelas is often the origin of erysipelas in other patients; but under certain circumstances it may cause an entirely different type of disease. Nurses suffering from erysipelas have conveyed puerperal fever to lying-in women; and medical men and nurses have contracted erysipelas when in attendance upon cases of puerperal fever. Moreover, a comparison of the registered deaths from erysipelas, puerperal fever, and pyæmia, shows a sufficient correspondence to suggest that a relationship

between these diseases exists. Again, a true cutaneous erysipelas cannot be sharply separated from phlegmonous erysipelas ; nor can the latter disease be always separated from allied septic inflammations. When we discuss the pathology of erysipelas, we shall find that the micro-organism which is the cause of the disease is probably identical with that found in many other septic conditions. But, in spite of these considerations, the clinical symptoms of cutaneous erysipelas are sufficiently characteristic to justify us in treating it as a distinct disease.

Geographical distribution.—Erysipelas is met with in both hemispheres, but is less frequent in the tropics than in temperate or cold climates.

Season.—It is most common during the colder seasons of the year. In Great Britain the cases, as judged by the mortality returns, appear to bear an inverse ratio to the rainfall, and cold east winds are said to favour the disease.

Age and Sex.—There is no evidence that sex has any decided influence upon the incidence of the disease. With regard to age we may remark that 30 per cent. of the registered deaths occurred during the first year of life.

Dissemination.—Under certain circumstances erysipelas spreads very rapidly from one person to another. In former times, when a case of erysipelas was introduced into a surgical ward, almost all the patients contracted the disease ; but now, on account of the antiseptic treatment of wounds, such an event does not occur. Nevertheless patients suffering from erysipelas should be isolated ; for there are always in surgical wards certain cases in which strict antiseptic treatment cannot be carried out. In medical wards the disease does not generally spread ; for

here the condition favouring the entrance of the virus (*i.e.*, the presence of open wounds) does not prevail.

The virus may be conveyed through the air, or by a third person, infected instruments, and fomites. Conveyance through the air is the least common mode of infection.

As with other infectious diseases, it is often impossible to trace the source of the contagion; and this is the less surprising when we bear in mind what has already been said of the relation of erysipelas to other septic diseases.

Predisposing causes.—In addition to the presence of an open wound, there are certain other conditions which favour the occurrence of erysipelas. Persons living under bad hygienic conditions are more liable to be attacked, and so are patients suffering from Bright's disease, chronic alcoholism, and other chronic affections.

Period of Incubation.—This is generally from one to three or four days. In Fehleisen's inoculations of human subjects the period varied between fifteen and sixty-one hours.

Clinical History.—Cutaneous erysipelas, to which our description is limited, has been divided into two varieties—*idiopathic* and *traumatic*. In the latter variety the disease starts in the neighbourhood of a wound, and in the former in some part of the skin where no obvious wound exists. But it is probable that idiopathic erysipelas always starts in some slight abrasion of the skin, and thus can be placed in the same category as traumatic erysipelas. At any rate, there is no appreciable difference in the symptoms in the two cases.

Period of invasion.—The attack almost always begins quite suddenly with a rigor or vomiting. The temperature rapidly rises to 104° or even 106° Fahr., and is attended

with the usual symptoms of fever. As a rule, the rash appears very quickly after the commencement of the attack, but it is sometimes delayed for some days. The lymphatic glands in the neighbourhood of the affected part may be often observed to be tender and enlarged before the eruption appears.

The rash.—The rash of erysipelas is not analogous to that of such a disease as scarlet fever; for in the latter disease it is only a subsidiary symptom, while in the former it is the local reaction at the spot of invasion, corresponding in this respect to the exudation in diphtheria. In idiopathic erysipelas the rash often begins near the inner canthus of the eye or the ala of the nose, as a redness or purple hue of the skin attended by a burning sensation and tenderness. In traumatic erysipelas it begins near the edge of the wound. The redness quickly spreads, and the skin becomes tense, shining, and swollen. The swelling is most marked where the skin is loose, as on the eyelids or scrotum. The margin of the affected part always presents a well-defined, hard, raised edge, which can easily be felt by the finger. Vesicles, and even bullæ, frequently appear. Erysipelas of the face does not usually affect the chin, and does not often extend to the chest, but it may spread all over the head and neck. In erysipelas of the limbs the rash may spread all over the body, but as it advances it subsides in the part previously attacked. It seldom starts in two distinct places. The duration of the rash is variable. As a rule, it lasts about seven days, but in some cases it may continue to wander about the body for several weeks. As it subsides the skin becomes flaccid and shrivelled, the vesicles dry up, and desquamation of the affected part occurs.

Course of the disease.—The temperature, which has risen rapidly, commonly remains at about 104° or 105° Fahr. until the crisis. Even in severe cases, however, the temperature during the whole of the attack may be only a fraction of a degree above normal. In prolonged cases

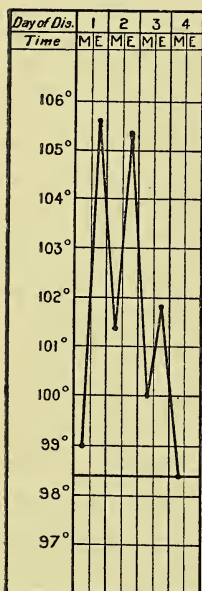


CHART Z.

Erysipelas arising in a scalp wound; no trace of the rash to be seen after the third day.

the temperature is very irregular. The pulse and respirations are increased with the temperature. In addition to the usual symptoms of fever there is frequently delirium of an active character. Continuous vomiting may be a troublesome symptom, and diarrhoea is common. Albuminuria is generally present, and in some cases blood and casts appear in the urine.

The duration of the attack is usually from six to eight days, but it may be prolonged for many weeks. The usual termination is by crisis, the temperature rapidly falling to normal. As the temperature falls the rash disappears, and the other symptoms subside. In fatal cases the patient falls into a low typhoid condition for some days preceding death.

Complications.—*Sloughing* of the affected skin may result, and subcutaneous *abscesses* may occur. If the scalp is affected the *hair* frequently falls off. Erysipelas of the neck may extend to the *larynx* and cause obstruction, necessitating the performance of tracheotomy. *Pneumonia*, *pleurisy*, and *peritonitis* are occasional complications. *Pyæmia* is rare, and so also is *meningitis*.

Sequelæ.—Recurrent attacks may lead to permanent œdema (elephantiasis).

Erysipelas sometimes causes a disappearance of chronic

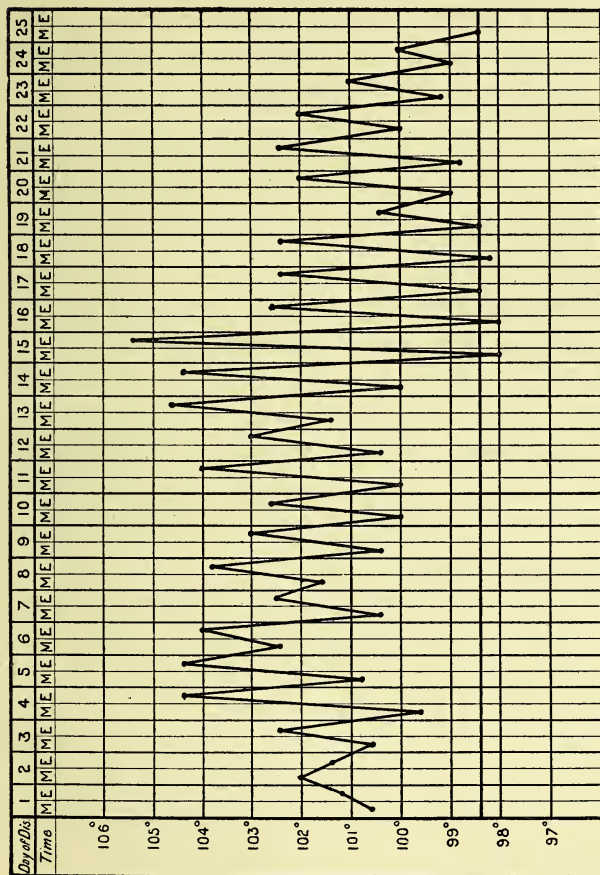


CHART Z2.

Temperature chart of a case of wandering and recurrent erysipelas starting from a wound of the left side of the neck, and affecting chiefly the head, trunk, and left arm,

skin diseases, and a shrinking of sarcomatous and other tumours. Attempts have been made to cure these diseases by the inoculation of pure cultivations of the specific micro-organism, but the erysipelas thus set up has

sometimes been so severe that this method of treatment is now abandoned. More recently the toxins produced by the growth of the streptococcus and the bacillus prodigiosus have been used for the treatment of sarcomata. A few minims injected into the tumour cause a local inflammation, pyrexia, and constitutional disturbances. In some cases a permanent cure appears to have been effected by this method of treatment.

Protection.—One attack protects only for a short time. Indeed, patients who have once been attacked are more liable to future attacks.

Relapses are of frequent occurrence.

Infectivity lasts from the commencement of the attack until at least a week after the disappearance of the cutaneous inflammation, or, in the case of a wound, after this has returned to a healthy state.

Morbid Anatomy.—Sections through the affected skin show the usual signs of inflammation. The lymph spaces, especially at the margin of the affected part, are filled with streptococci.

The *streptococcus erysipelatis*.—This micro-organism consists of cocci generally arranged in chains. Diplococci and irregular masses of cocci are often met with. In the human body the chains are short, but in artificial cultivations long twisted chains are formed. It stains with the usual aniline dyes, and by Gram's method.

It grows best at the body temperature, but will also grow at the usual temperature of the air. Gelatine, agar, broth, and blood-serum are all good media for cultivation. The colonies are characterised by their small size and by their transparency. They do not liquefy gelatine.

Pathology.—It is quite certain that the streptococcus is the cause of erysipelas. It is constantly present in

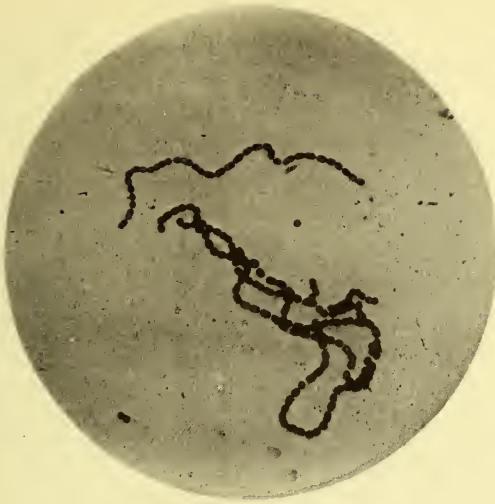


Fig. 1.—*Streptococcus Erysipelatis*. Broth cultivation. Gram. $\times 1000$.

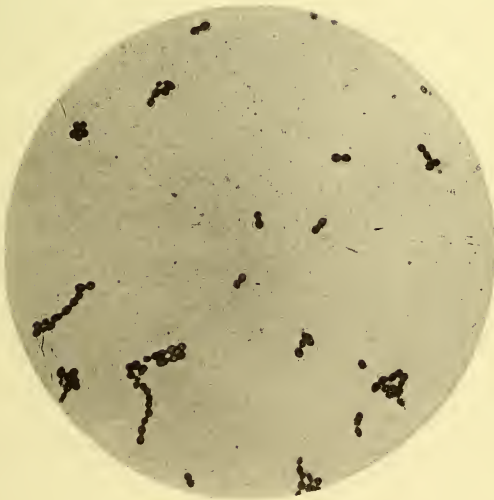


Fig. 2.—*Streptococcus Erysipelatis*. Agar cultivation. Gram. $\times 1000$.

the affected skin, and inoculations of pure cultivations in the human subject produce a typical erysipelas. Such inoculations have been made with the object of curing malignant tumours.

A similar streptococcus, the streptococcus pyogenes, is the cause of various septic affections, such as puerperal fever, septicæmia, and phlegmonous erysipelas. There has been much discussion as to the relationship of this streptococcus to the streptococcus erysipelatis. Various differences have been described in morphology, mode of growth, and pathogenic effects upon animals. It is beyond the scope of this book to enter into a discussion upon these interesting questions. Suffice it to say that the balance of evidence is in favour of the identity of the streptococcus erysipelatis with the streptococcus pyogenes. Assuming that this view is correct, we can readily understand how a case of erysipelas can give rise to puerperal fever or some other septic infection in another patient.

The difference in the symptoms in the various streptococcal infections is partly due to a varying virulence of the streptococcus, and partly to the fact that the characteristic type of the infection is masked by the local effects of the inflammation. In tuberculosis we observe the same variability of symptoms according to the part attacked. The symptoms of tubercle of the meninges are quite different from those of tubercle of the peritoneum or of the synovial membranes. Experiments upon animals show that streptococci obtained from various sources in the human subject differ in virulence. As a rule, laboratory animals are not very susceptible, and subcutaneous inoculation with streptococci cultivated directly from the human subject frequently causes only a local inflammation, ending in recovery. But by repeated

passages the virulence of the streptococcus can be so intensified that a minute quantity injected under the skin causes death from a general septicæmia.

Animals can be immunised by inoculations firstly with attenuated, and subsequently with virulent cultivations. The blood serum of immunised animals will protect other animals against infection. Horses have been immunised, and the blood serum used for the treatment of erysipelas and other streptococcal infections in the human subject.

Diagnosis.—Traumatic erysipelas can readily be recognised by the rash with the well-defined raised margin, and the accompanying constitutional symptoms.

Idiopathic erysipelas is not always easy to diagnose. It may be mistaken for erythema, and when on the face for commencing herpes. The constitutional symptoms and the special characters of the rash soon enable a correct diagnosis to be made. In a doubtful case a cultivation of the streptococcus can be made by puncturing the margin of the skin and inoculating tubes of agar or broth.

Prognosis.—The prognosis is favourable in previously healthy adults. In infants and old people the prognosis is bad ; and it is especially unfavourable in patients with chronic kidney disease, and in alcoholics. It should be remembered that death may result from implication of the larynx.

In all cases severe delirium and the typhoid condition are grave symptoms.

Treatment.—The patient should be isolated. The diet should be restricted to such articles of food as milk, eggs, and beef-tea, as long as the temperature is raised. Stimulants should be given as required. At the commencement of the attack a brisk purge of calomel should

be given, and subsequently iron and quinine should be administered. Unless the diarrhœa is excessive it is better not to check it.

The surface of the affected skin should be dusted with starch and oxide of zinc powder. Painting the margin of the inflamed skin with nitrate of silver sometimes appears to stop the advance of the inflammation.

In traumatic erysipelas the wound should be treated on antiseptic principles.

The results obtained by treatment with the antistrep-tococcal serum are sufficiently encouraging to justify the prediction that this remedy will ultimately prove to be of great value.

CHAPTER XX.

ANTHRAX. MALIGNANT PUSTULE. WOOL-SORTER'S DISEASE. SPLENIC FEVER.

THIS is a form of septicæmia caused by the anthrax bacillus, and characterised by the formation of a serous or hæmorrhagic exudation at the seat of inoculation. It is transmitted to the human subject by direct or indirect contact with infected animals.

Etiology.—Epidemics of anthrax occur among sheep, cattle, horses, goats, deer, and other animals. The disease is widely distributed over the world, and is especially prevalent in Russia, where it causes great ravages. In England only some three hundred to six hundred cases occur annually, and in the United States it is decidedly rare.

The disease is spread in the following manner. The blood of infected animals contains the bacilli; and when the animals are slaughtered the soil becomes infected with the blood which is spilt. Infection of the soil also occurs from the intestinal evacuations and from discharges from the mouth and nose. The bacilli themselves are readily destroyed by putrefactive bacteria, but in the presence of oxygen they quickly form spores, which are highly resistant. Consequently the soil remains infective for

a long time, and animals grazing upon the infected pastures become attacked. If the carcasses of infected animals are buried immediately after death, spores are not formed, and the bacilli are destroyed by the process of putrefaction. By adopting this procedure a fertile source of infection is removed. Anthrax can also be conveyed from infected to healthy animals by means of flies.

This disease is communicated to the human subject in several ways. The most common method is by means of the hides or wool of infected animals. The bacilli in the blood adherent to the skin become converted, after the death of the animal, into spores, which, as has already been stated, are highly resistant to destructive agents. In England, the wool-sorters of Bradford and the tanners of Bermondsey are frequently infected by wool and hides, imported from districts where the disease is prevalent. Slaughtermen and butchers are sometimes inoculated with the blood of infected animals, and instances have been recorded where the disease has been contracted by the ingestion of infected meat.

The virus may enter the body through some abrasion of the skin—*external anthrax*, or through the respiratory or intestinal tract—*internal anthrax*. External anthrax is most common among tanners, and internal anthrax among wool-sorters. In the former case inoculation occurs by direct contact with the hides, and in the latter by inhaling or swallowing dust containing the spores.

Anthrax in the human subject is a comparatively rare disease in England, as will be seen from the following table taken from the Registrar-General's Report for 1894 :—

Registered deaths from anthrax and splenic fever in England and Wales (including London) for the years 1875—1894.

1875 ...	5	1882 ...	15	1889 ...	7
1876 ...	4	1883 ...	8	1890 ...	4
1877 ...	10	1884 ...	18	1891 ...	3
1878 ...	14	1885 ...	11	1892 ...	6
1879 ...	12	1886 ...	11	1893 ...	7
1880 ...	18	1887 ...	11	1894 ...	10
1881 ...	9	1888 ...	12		

The fatality of the disease is different in external and internal anthrax.

Of 117 cases of external anthrax collected by Dr. Hamer 24 died, a fatality of 20·5 per cent.

Of 23 cases of internal anthrax collected by Mr. Spear 19 died, a fatality of 82·6 per cent. The statistics of internal anthrax must, however, be accepted with caution, inasmuch as it is probable that mild cases were not recognised and were consequently not included in these figures.

EXTERNAL ANTHRAX OR MALIGNANT PUSTULE.

In this form of the disease the virus enters through some abrasion of the skin.

Clinical History.—The exposed parts of the body—the face, neck, or fore-arms—are most liable to be affected. A small, red, itching papule forms at the seat of inoculation, and is quickly converted into a vesicle. The vesicle either dries up or its roof is rubbed off, and a depressed black eschar is produced. Around the eschar a ring of vesicles frequently develops, but this is not always the case. The surrounding tissue becomes œdematous and of a bright red or livid hue, and with the central eschar and ring of vesicles presents a very characteristic appearance. Pain is conspicuous by its

absence, but complaint is usually made of a gnawing or pricking sensation. The lymphatic glands in the neighbourhood become enlarged and somewhat tender.

In some cases no constitutional symptoms develop, the vesicles dry up, the œdema subsides, the eschar separates, and recovery ensues. But as a rule the œdema increases to an enormous extent and constitutional symptoms arise. The onset of these symptoms may occur at any time from a few hours to eight or nine days after the first appearance of the local lesion. The most common period is four or five days. The principal symptoms are shivering or rigors, irregular pyrexia (101° — 104° Fahr.), a frequent and weak pulse, vomiting, diarrhœa, lividity, and profuse perspiration. Vomiting and diarrhœa are especially marked if secondary lesions occur in the stomach and intestines. In the latter case the motions may contain blood. When secondary lesions arise in the brain or spinal cord, delirium, convulsions, paralysis, or coma develops. In cases of anthrax of the neck and face the œdema may spread to the larynx or into the mediastinum, producing urgent dyspnœa.

Death may be due to collapse as a result of a general infection, or more frequently to implication of the larynx, or a secondary cerebral lesion. Recovery after excision of the pustule may ensue, even if the constitutional symptoms have been marked. Cases of recovery have been recorded where a general infection has been proved by the presence of the bacilli, either in the blood of the general circulation or in the various excretions.

Mild cases.—In addition to the cases in which the "pustule" becomes fully developed but resolves without the appearance of constitutional symptoms, there are other cases in which it remains undeveloped. This form

occurs especially on the hands. The lesion starts in the usual way as a small itching papule, which either resolves at once, or after the discharge of a serous exudation and the formation of œdema in the surrounding tissue. Mr. Spear states that most of the wool-sorters at Bradford are affected in this way at some time or other. It must be mentioned that no absolute bacteriological proof of the nature of these cases has been brought forward.

INTERNAL ANTHRAX.

In this form of the disease the virus enters the body either through the respiratory or the digestive tract. It is probable that it usually enters through the respiratory tract.

The **incubation period** is difficult to determine. Mr. Spear quotes a case in which it was probably not longer than two days, but he gives reasons for believing that the germs may remain latent for weeks before giving rise to symptoms.

Clinical History.—Mr. Spear divides the attack into two periods, the prodromal stage and the stage of full development.

The *prodromal stage* lasts as a rule two to five or six days, but it may be only of a few hours' duration. The principal symptoms are shivering, pains in the limbs, mental depression, dyspnœa, and a sense of constriction in the chest. There may be, in addition, vomiting, nausea, headache, dizziness, formication in the limbs, or a staggering gait. Distinct remission of the symptoms is common.

The *stage of full development* usually comes on quite suddenly. In the most acute cases the respirations are increased to 30 or 40 per minute, the pulse runs up to

120 or 150 and becomes weak and compressible, there may be pyrexia, and the patient dies in a condition of collapse in twelve to forty-eight hours after the first onset of serious symptoms.

In the majority of cases the course is more prolonged. The temperature rises to 104° or 106°, the prodromal symptoms become more pronounced, and the patient dies in a few days. There is sometimes distinct evidence of implication of the lung in the expectoration of a frothy blood-stained fluid. Gastro-intestinal symptoms (vomiting and diarrhoea with bloody evacuations) may be a prominent feature. Again, cerebral symptoms (delirium, convulsions, and coma) are not uncommon. In some cases there is a remission of all the symptoms; nevertheless the patient ultimately dies, either from collapse occurring quite unexpectedly, or with symptoms indicating an affection of the central nervous system, or from dyspnoea due to oedema of the mediastinum.

When recovery ensues convalescence is protracted, and there are often for a long time muscular tremor and cramp.

Mild cases apparently occur, in which the disease does not develop beyond the prodromal stage.

Relapses, according to Mr. Spear, are sometimes met with.

Morbid Anatomy.—Sections through a malignant pustule show the presence of a serous exudation in the connective tissue. The eschar consists of necrosed tissue. A distinct pus formation is rare. In the exudation anthrax bacilli are present in large numbers. A secondary invasion of the pustule with pyogenic cocci is common.

When death has taken place from *external anthrax* a post-mortem examination almost always reveals secondary

lesions in various parts of the body, in addition to œdema at the seat of inoculation. In the stomach and intestines the lesions take the form of patches of gelatinous or sanguineous infiltration in the submucous tissue, with sometimes a black slough in the mucous membrane. In the central nervous system the lesions consist of hæmorrhagic infiltrations into the meninges, or into the substance of the brain or cord. Œdema spreading from the neck to the larynx or mediastinum is common. Besides these lesions there may be any of the conditions found in death from internal anthrax.

A post-mortem examination of cases of *internal anthrax* usually reveals the presence of swelling of the mucous membrane of the lower part of the trachea and the bronchi, which contain blood-stained fluid. The bronchial glands are enlarged and often hæmorrhagic, and there is frequently serous exudation in the mediastinal tissue. A serous exudation may also be found in the pleural or pericardial cavities. The lungs and other parts of the body may present hæmorrhages, and secondary lesions similar to those described in external anthrax may be found in the gastro-intestinal tract or in the central nervous system. The spleen is sometimes enlarged and soft, and sometimes of normal size and consistence.

In both forms of the disease anthrax bacilli are always found in the local lesions, and they are sometimes diffused all through the body.

The *anthrax bacillus* is a non-motile rod-shaped micro-organism, which often grows out into long jointed threads matted together like the strands of a rope. It is not decolorised when stained by Gram's method. In the presence of oxygen large oval spores are formed. By cultivation in media containing carbolic acid sporeless



Fig. 1.—Anthrax bacillus.
Cover glass impression.
Agar plate. $\times 500$.

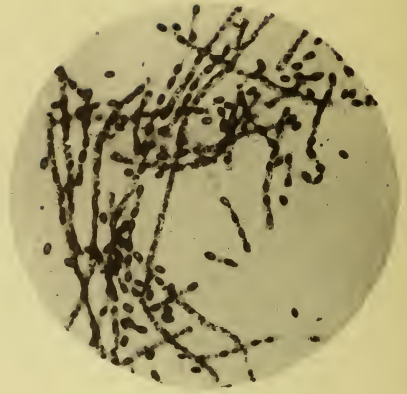


Fig. 2.—Anthrax bacillus.
Spores.
Moeller's method. $\times 1000$.



Fig. 3.—Anthrax bacillus.
Exudation from malignant pustule in man.
Gram. $\times 1000$.

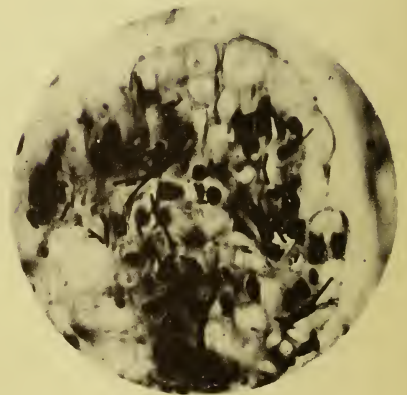


Fig. 4.—Anthrax bacillus.
Glomerulus of kidney of mouse.
Picro-carmin, Gram-Weigert. $\times 1000$.

varieties of the bacillus can be obtained. The bacillus will grow readily in a large number of media and at the ordinary temperature of the air. It grows best in presence of oxygen, but it is not a strict aerobe. On gelatine plates the colonies have an irregular outline, and under a low power of the microscope are found to consist of felted masses of threads. The surrounding gelatine becomes liquefied. A stab cultivation in gelatine has often, though not always, a characteristic appearance. Lateral threadlike ramifications pass out from the line of puncture into the medium, so that the growth has the appearance of a root with lateral rootlets. After a time the gelatine liquefies, beginning at the surface, and the growth sinks as a horizontal white layer separating the solid from the liquefied gelatine. On the surface of agar the growth presents a felted appearance, on potato it forms a pasty white layer, and in broth a felted mass falls to the bottom of the tube.

Pathology.—There is abundant evidence that the anthrax bacillus is the cause of the disease. A large number of animals are susceptible to inoculation with pure cultivations. Mice, rabbits, and guinea-pigs develop a local œdema and die of a general infection when inoculated in the subcutaneous tissue. In these animals the spleen is always found to be enlarged, hence the term splenic fever. Sheep and cattle are more susceptible to ingestion of spores into the alimentary system than to subcutaneous inoculation. White rats and Algerian sheep enjoy immunity. In dogs a successful inoculation leads to the formation of a typical “malignant pustule,” generally ending in recovery.

The virulence of the bacillus can be attenuated by cultivation at high temperatures, or by the addition of

carbolic acid to the media. Inoculation with attenuated cultivations gives rise to a mild form of the disease, and after recovery the animals are immune to inoculation with virulent cultivations. "Vaccination" of sheep with attenuated cultivations has been successfully practised. Dr. Sidney Martin has isolated from cultivations and from the viscera albumoses and an alkaloid, which when injected produce the symptoms of the disease.

In malignant pustule in the human subject the virus enters through an abrasion of the skin and multiplies at the spot of inoculation. It may be destroyed, recovery ensuing; or it may be carried to various parts of the body, producing "pustules" in the internal organs; or it may cause a general infection of the blood. In wool-sorter's disease the frequent swelling of the submucous tissue of the trachea and bronchi, with the presence of bacilli in these parts, and the implication of the bronchial glands, are evidence of the entrance of the virus through the respiratory tract.

Diagnosis.—The diagnosis of a typical case of external anthrax is easy, the black eschar, the ring of vesicles, and the surrounding œdema being quite characteristic. Absence of pain serves to distinguish it from other forms of acute infective wounds. Atypical pustules are difficult to diagnose, but a bacteriological or even a simple microscopical examination removes all doubt as to the true nature of the case.

Internal anthrax offers much greater difficulties. A history of exposure to contagion must often be relied upon as the chief means of forming an opinion. The specific bacillus may be found in the blood-stained expectoration or alvine discharge, or, in late stages of the disease, in the blood of the general circulation.

In making a bacteriological examination a little of the fluid from the vesicle should be spread upon a cover-glass, and stained either by Gram's method or with carbolic methylene blue. A rapid diagnosis can be made with moderate certainty in this way. More accurate results can be obtained by making plate cultivations in gelatine, or by the inoculation of mice or guinea-pigs.

Prognosis.—In external anthrax the prognosis depends to a large extent upon the early recognition and excision of the local lesion. Of the 98 cases collected by Dr. Hamer in which excision was practised 12 died, a fatality of 12·2 per cent.; while of 18 treated in other ways 11 died, a fatality of 61·1 per cent. The region affected is of importance. When the neck is the seat of inoculation the œdema may spread to the larynx or the mediastinum. Many patients die in this manner from asphyxia. In all cases the more extensive the œdema, the more unfavourable is the prognosis. When the "pustule" is ill developed the prognosis is good. The occurrence of constitutional symptoms, and especially the appearance of symptoms indicating secondary lesions in the central nervous system, are of grave import. It must, however, be remembered that patients not infrequently recover even when constitutional symptoms have become well developed.

The prognosis of internal is much more serious than that of external anthrax. The fatality has been given at 82·6 per cent, and although this is probably too high, it nevertheless represents the fatality of cases in which the symptoms are sufficiently developed for a definite diagnosis to be made. In individual cases special attention should be paid to the heart. Any indication of failure should arouse the gravest fear. As with external anthrax, the implication of the central nervous system is most serious.

Treatment.—In cases of external anthrax the pustule should be at once excised and potassa fusa or chloride of zinc paste (1 in 3) applied, or the surrounding tissue seared with a thermo-cautery. Mr. Davies-Colley has used with success a treatment recommended by Dr. Muskett, consisting in excision and the external application of ipecacuanha powder, combined with the internal administration of the same drug.

Constitutional symptoms should be treated upon general principles.

APPENDIX I.

Carbolic Methylene Blue Solution.

Methylene blue	1·5 g.
Absolute alcohol	10 c.c.
Carbolic acid	5 g.
Water	100 c.c.

Carbolic Fuchsin Solution.

Fuchsin	1 g.
Absolute alcohol	10 c.c.
Carbolic acid	5 g.
Water	100 c.c.

To make Aniline Water Gentian Violet Solution.

Aniline oil is shaken with distilled water and filtered. To the filtrate a saturated alcoholic solution of gentian violet is added until the mixture becomes opalescent.

To stain Flagella (Pittfield's Method).

Two solutions are required:—

- A.—Tannic acid. 1 g.
 Water 10 c.c.
- B.—Saturated aqueous solution of alum . . . 10 c.c.
 Saturated alcoholic solution of gentian violet . 1 c.c.

Immediately before use mix equal parts of A and B. Put a little of the mixture on the cover-glass, and heat over a spirit-lamp until the liquid boils. After the end of a minute wash in water, dry, and mount.

APPENDIX II.

THE REGULATIONS OF THE METROPOLITAN ASYLUMS BOARD RELATING TO THE REMOVAL OF PATIENTS SUFFERING FROM INFECTIOUS DISEASES.

Applications for Ambulance Carriages for the Removal of Patients must be made as follows :—

On WEEK DAYS, between 9 a.m. and 8 p.m., to the CHIEF OFFICES (Ambulance Department), Norfolk House, Norfolk Street, Strand, W.C.

TELEGRAPHIC ADDRESS :—Asylums Board, London.

TELEPHONE NUMBERS :—2,587 and 35,354.

*** Applications in the latter part of the day must be dispatched in time to reach the Offices before 8 p.m.*

At NIGHT, between 8 p.m. and 9 a.m., and on Sundays, Christmas Day, and Good Friday, to the AMBULANCE STATIONS :—

Eastern Ambulance Station,
Brooksby's Walk, Homerton, N.E.

Western Ambulance Station,
Seagrave Road, Fulham (near West Brompton
Railway Station, S.W.).

South-Eastern Ambulance Station,
New Cross Road (near Old Kent Road Railway
Station, S.E.).

Brook Ambulance Station,
Shooter's Hill, Kent.

REMOVAL TO THE BOARD'S HOSPITALS.

- (a) Only persons suffering from Small-pox, Fevers, or Diphtheria are admitted into the Board's Hospitals.
- (b) Every application must state the name, age, and full address of the patient, from what disease suffering, and in cases of fever the particular kind of fever; and also the name of the person making the application.
- (c) Unless a Medical Certificate of the nature of the disease be handed to the Ambulance Nurse the patient will not be removed.
- (d) Patients should leave all valuables, money, etc., and all outside clothing, at home; should wear body linen only, and be wrapped in the blankets provided for the purpose.
- (e) The Ambulance Nurse will leave at the house from which the patient is removed a notice stating the Hospital to which the patient is to be taken, and a copy of the regulations as to visiting, etc.

N.B.—In any case of failure to remove on the day of application, in consequence of pressure on the accommodation in the Board's Hospitals, the application must be renewed every morning so long as the patient continues in a fit state for removal and removal is desired. Each application must contain a statement of the circumstances which render the removal urgent.

Similar applications must be made in respect of patients taken from General Hospitals or other places to their homes, owing to there being no vacant bed available in the Board's Hospitals.

Forms of application for the use of Public Officials may be obtained upon application to the Clerk to the Board.

CONVEYANCE TO OTHER PLACES.

- (a) Persons suffering from any dangerous Infectious Disease* may be conveyed by Ambulance to places other than the Board's Hospitals.
- (b) Every application for an Ambulance must state—
 - (i) Name, sex, and age of patient.
 - (ii) Description of disease, and, in the case of fever, the particular kind of fever.

* Dangerous Infectious Diseases include the following:—Small-pox, Cholera, Diphtheria, Membranous Croup, Erysipelas, Scarlatina or Scarlet Fever, Typhus, Typhoid, Enteric, Relapsing, Continued, and Puerperal Fevers. The Ambulance Committee have also authorised the use of their Ambulances for Measles and Chicken-pox.

- (iii) Full address *from* which the patient is to be conveyed.
- (iv) Full address *to* which the patient is to be conveyed.
(Arrangements for the reception of the patient must be made before application for the Ambulance.)
- (c) The patient must be provided with a Medical Certificate of the nature of the disease, to be handed to the Driver of the Ambulance.
- (d) The charge for the hire of the Ambulance, including (when the patient is over ten years of age) the services of a male attendant, is 5s. This amount must be paid to the Driver, who will give an official receipt for the same.
- (e) One person only will be allowed to accompany the patient, and such person may be conveyed back to the place from which the patient was conveyed. If desired, a Nurse will be supplied at an additional charge of 2s. 6d. for her services.
- (f) The Ambulances may be sent outside the Metropolitan district only by special sanction of the Ambulance Committee or of the Clerk to the Board, and in such cases an extra charge will be made of 1s. for every mile outside the Metropolitan area.

The Drivers of the Board's Ambulances are not allowed to loiter on their journeys or to stop for refreshments on pain of instant dismissal. It is particularly requested that any breach of this regulation, or any neglect or incivility on the part of the Drivers, Nurses, or Attendants, may be immediately reported to the Clerk to the Board.

The Servants of the Board are forbidden to accept any gratuities or refreshments.

N.B.—PENALTY.—By Section 70 of the Public Health (London) Act, 1891, it is enacted that "It shall not be lawful for any owner or driver of a public conveyance knowingly to convey, or for any other person knowingly to place in any public conveyance, a person suffering from any dangerous infectious disease, or for a person suffering from any such disease to enter any public conveyance, and if he does so he shall be liable to a fine not exceeding TEN POUNDS."

APPENDIX III.

TABLE SHOWING LENGTH OF INCUBATION PERIOD, DATE OF APPEARANCE OF ERUPTION, ETC., OF THE MOST COMMON INFECTIOUS DISEASES.

Disease.	Length of Incubation Period.	Day of Disease upon which Eruption appears.	Length of Time of Isolation of Patient.	Length of Quarantine Period.	Protection afforded.
1. Influenza ..	2 or 3 days; may be a few hours	No eruption ..	—	—	Relapses and second attacks common.
2. Diphtheria..	2 days (1 to 5) ..	No eruption ..	As long as diphtheria bacilli are present; failing a bacteriological examination, for 3 weeks from disappearance of exudation	7 days	Relapses and second attacks not uncommon.
3. Scarlet Fever ..	3 days (a few hours to 5 days)	Usually within 24 hours	For at least 6 weeks from appearance of rash; beyond 6 weeks it is advisable to isolate until desquamation is complete or any chronic discharge has ceased	7 days	Relapses uncommon; second attacks not uncommon.
4. Whooping Cough..	5 to 14 days ..	No eruption ..	Till the disappearance of the whoop	15 days	Relapses and second attacks are rare.
5. Measles (Morbilli)	7 to 18 days ..	Usually on 4th day (1st to 6th)	21 days from appearance of rash	21 days	Relapses rare; second attacks infrequent.
6. Typhus Fever ..	12 days (5 to 14) ..	Usually on 5th day (4th to 8th)	4 weeks from commencement of illness	15 days	Relapses excessively rare; second attacks rare.
7. Small-pox ..	12 days (10 to 14) ..	3rd day (occasionally earlier or later)	Till all scabs have separated ..	15 days	Relapses do not occur; second attacks rare.
8. Rubella ..	14 days (11 to 18) ..	1st day ..	10 days from appearance of rash	21 days	Relapses do not occur; second attacks occasionally met with.
9. Enteric Fever ..	14 days (occasionally less)	4th to 7th day (occasionally before 4th)	—	—	Relapses common; second attacks rare.
10. Chicken-pox ..	12 to 19 days ..	1st day (occasionally 2nd)	Till all scabs have separated ..	21 days	Relapses and second attacks are very rare.
11. Mumps ..	21 days (14 to 25) ..	No eruption ..	3 weeks from onset of parotitis	26 days	Relapses and second attacks uncommon.

The diseases are arranged in order of shortness of incubation period. The figures in brackets in the second and third columns indicate the extremes of the incubation periods and the earliest and latest dates of appearance of eruption. The incubation periods of whooping cough, measles, and chicken-pox are very variable. By Quarantine Period is meant the length of time during which a susceptible person, who has been exposed to infection, is to be isolated. It is advisable to make this period a day or two longer than the longest known incubation-period of any particular disease.

APPENDIX IV.

THE ANTITOXINE TREATMENT OF DIPHTHERIA.

Since the account given in the text has passed through the press, the Report of the Medical Superintendents of the Fever Hospitals of the Metropolitan Asylums Board on the use of antitoxic serum in the treatment of diphtheria has been published.

The number of antitoxine-treated cases dealt with in the Report is 2,182 (see Table I.).

TABLE I.—CASES TREATED WITH ANTITOXINE DURING 1895, SHOWING DAY OF DISEASE ON WHICH THE TREATMENT WAS COMMENCED.

Day of Disease.	1st.		2nd.		3rd.		4th.		5th and after.		Total.		Mortality per cent.
Ages.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	
Under 1	1	0	10	5	8	6	5	0	13	7	37	18	48.6
1 to 2	10	2	37	13	40	17	30	20	65	42	182	94	51.6
2 „ 3	9	0	38	5	42	18	39	16	59	26	187	65	34.7
3 „ 4	16	2	46	8	64	17	61	25	123	52	310	104	33.6
4 „ 5	10	0	54	8	50	13	82	38	101	39	297	98	32.9
5 „ 10	30	0	145	18	170	40	159	45	312	93	816	196	24.0
10 „ 15	10	0	53	2	44	6	46	4	74	19	227	31	13.6
15 „ 20	0	0	7	1	12	1	12	2	19	2	50	6	12.0
20 and upwards }	0	0	13	0	19	0	19	0	25	3	76	3	3.9
Total	86	4	403	60	449	118	453	150	791	283	2182	615	28.1
Mortality } per cent. }		4.6		14.8		26.2		33.1		35.7		28.1	

} 37.4

For comparison with these cases the 3,042 cases under treatment during the year 1894, immediately before the introduction of the antitoxine treatment, may be taken. (See Table II.)

TABLE II.—CASES OF DIPHTHERIA UNDER TREATMENT DURING 1894, IMMEDIATELY BEFORE THE ANTITOXINE TREATMENT WAS COMMENCED

(Day of Disease = day of disease on which patient was admitted to hospital.)

Day of Disease.	1st.		2nd.		3rd.		4th.		5th nd after.		Total		Mortality per cent.
Ages.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	Cases.	Dths.	
Under 1	0	0	10	7	4	1	11	6	15	11	40	25	62.5
1 to 2	10	5	42	29	47	25	39	25	66	42	204	126	61.7
2 „ 3	16	7	55	27	51	22	44	26	91	50	257	132	51.3
3 „ 4	17	6	58	26	70	37	71	37	126	55	342	161	47.0
4 „ 5	15	4	51	18	71	26	47	14	144	50	328	112	34.1
5 „ 10	53	6	179	34	215	71	208	60	420	109	1075	280	26.0
10 „ 15	12	2	85	4	79	8	59	5	128	22	363	41	11.2
15 „ 20	6	0	23	0	33	1	28	0	70	6	160	7	4.3
20 and upwards }	4	0	36	1	82	1	59	6	92	10	273	18	6.5
Total	133	30	539	146	652	192	566	179	1152	355	3042	902	29.6
Mortality } per cent. }		22.5		27.0		29.4		31.6		30.8		29.6	

47.4

Two striking facts are apparent in these tables. The first is that in the antitoxine cases the mortality per cent. in those coming under treatment on the first day was 4.6, and on the second day 14.3, against 22.5 and 27.0 for the non-antitoxine cases. “The aggregate mortality among cases which came under treatment on the first three days of illness was 19.4 per cent. in the antitoxine cases, and 27.7 per cent. in those treated by other methods in 1894.” The second is, that for every age-group under ten years of age the mortality was less among the antitoxine than the non-antitoxine cases.

Above ten years of age the mortality was higher in the antitoxine than the non-antitoxine cases: 11.3 per cent. for the former, 8.2 per cent. for the latter. The Report, however, makes a statement that throws some light on this difference—namely, that as a rule the mild cases were not treated with antitoxine. Now the proportion of mild cases among adults is always high, and consequently the non-antitoxine series would include a larger number of mild cases than the antitoxine series.

The following table (Table III.) gives the results of tracheotomy cases for the non-antitoxine cases of 1894 and the antitoxine cases of 1895:—

TABLE III.

Ages.	1894 (non-antitoxine).			1895 (antitoxine).		
	Cases.	Deaths.	Mortality per cent.	Cases.	Deaths.	Mortality per cent.
Under 1	5	4	80.0	4	3	75.0
1 to 2	33	29	87.8	48	34	70.8
2 „ 3	46	35	76.0	33	16	48.4
3 „ 4	53	33	62.2	52	25	48.0
4 „ 5	45	32	71.1	40	17	42.5
5 „ 10	75	47	62.6	46	16	34.7
10 „ 15	1	1	100.0	2	2	100.0
15 and upwards.	3	3	100.0	0	0	0.0
Total	261	184	70.4	225	113	50.2

From this table it is seen that, while the recovery-rate (29.6 per cent.) was good in 1894, it was very much better (49.8 per cent.) in the antitoxine cases of 1895.

In many cases the antitoxine treatment did away with the necessity for performing tracheotomy. The Report states that whereas in 1894 56.0 per cent. of the cases of laryngeal diphtheria required tracheotomy, in 1895 the percentage was 45.3. Further, whereas during 1894, in 3.8 per cent. of the cases the larynx became involved after the patient's admission to hospital, in 1895 the percentage of such cases fell to 0.6.

The following table shows the percentage of the recognised complications of diphtheria in the two series of cases:—

TABLE IV.

Complications.	Col. 1. 1894. Non-antitoxine Cases.	Col. 2. 1895. Antitoxine Cases.	Col. 3. 1895. Non-antitoxine Cases.
Albuminuria	24.1	40.9	28.6
Nephritis	1.2	2.0	1.0
Paralysis (various)	13.2	23.2	16.7
Pneumonia (lobar)	0.3	0.8	1.1
„ (lobular)	1.6	3.6	1.2
Relapse of Disease	0.9	1.4	1.2

From this table it appears that the percentage incidence of all the complications is higher among the antitoxine than the non-antitoxine cases, especially as regards albuminuria, paralysis, and lobular

pneumonia. The writers of the Report make the following observations upon this fact:—"It is impossible to assign this increase to the operation of any one cause. That antitoxine alone cannot be responsible for it is shown by reference" to the above table (compare col. 1 with col. 3); "but the following considerations indicate some of the factors which may possibly have contributed to this result:—

- (i) "If any method of treatment for diphtheria is more efficacious than another in tiding the patient over the acute stage of the disease, it is only to be expected that the comparative incidence of complications amongst cases so treated would rise. In most fatal attacks death occurs fairly early, and the longer life is prolonged the greater is the likelihood that some complications will arise."
- (ii) "It is possible that the average type of disease in 1895 was more conducive to the development of certain complications. This is clearly suggested by the table which refers to the non-antitoxine cases of 1894 and 1895." (See Table IV., col. 1 and 3).
- "It is seen that, with the exception of nephritis and lobular pneumonia, the incidence of all the regular complications of diphtheria was greater amongst the non-antitoxine cases of 1895 (for the most part the mild ones) than amongst those of all degrees of severity in 1894."
- (iii) "It is possible that those concerned in taking the notes during 1895 have been more careful in recording the slighter manifestations of the various complications than in previous years; for certainly the introduction of the antitoxic treatment has led to a deepening of the interest attaching to the clinical study of diphtheria."

In Table V. is given the percentage incidence of the complications caused by the antitoxine treatment.

TABLE V.

Complications.	Percentage Incidence.
Rash	45'9
Joint-pains	4'7
Pyrexia, with or without rash or pains	29'6
Abscess at site of injection	2'3

"From this table it will be seen that a rash is the most common sequel. It usually takes the form of an urticaria, or a vivid, patchy

erythema, more or less covering the trunk and extremities, and is very similar to the eruptions of measles and septicæmia. It is sometimes scarlatiniform. It is often accompanied by pyrexia. This secondary fever, in some cases, persists for several days, and may be unaccompanied by any other obvious symptom : it has the effect of somewhat retarding convalescence, and no doubt in rare instances, in patients whose vitality has been lowered by a severe attack of diphtheria, may act prejudicially if it arise at a time when symptoms of cardiac failure are present. But the risk associated with this secondary pyrexia is very small compared with the benefit which follows the employment of antitoxine in the early acute stage of the disease. Other drugs of acknowledged usefulness are occasionally observed to give rise to symptoms which are the reverse of pleasant, and in this respect antitoxic serum is no exception."

"The joint-pains are rarely severe or accompanied by obvious effusion. They almost invariably pass off in the course of a few days, and apparently leave no ill effects."

The Report, which is signed by Drs. Gayton, MacCombie, Bruce, Caiger, Goodall, and Matthews, concludes with the statement : "We are further of the opinion that in antitoxic serum we possess a remedy of distinctly greater value in the treatment of diphtheria than any other with which we are acquainted."

There is a valuable appendix by Dr. Hume on the use of the serum in cases of post-scarlatinal diphtheria.

I N D E X .

Acme, 4.

Age-incidence : of chicken-pox, 230 ; of diphtheria, 112 ; of enteric fever, 294 ; of erysipelas, 331 ; of influenza, 254 ; of measles, 165 ; of mumps, 249 ; of relapsing fever, 280 ; of rubeola, 184 ; of scarlet fever, 58 ; of small-pox, 189 ; of typhus fever, 267 ; of whooping-cough, 242.

Albuminuria : in diphtheria, 118 ; in erysipelas, 334 ; in scarlet fever, 80 ; in small-pox, 203 ; in typhus fever, 273.

Anatomy, Morbid. See Morbid anatomy.

Anthrax, 340.

Antipyretics, 12, 104.

Antiseptics, 41.

Antitoxic treatment, 51, 160, Appendix IV.

Antitoxines, 31, 33.

Anti-vaccination, 228.

Anuria : in diphtheria, 118 ; in relapsing fever, 282.

Arthritis : in antitoxic treatment, 164, Appendix IV. ; in scarlet fever, 80.

Arthritis, suppurative, in scarlet fever, 81.

Bacilli, 17 : of anthrax, 346 ; coli, 318 ; of diphtheria, 137 ; of enteric fever, 317 ; Hoffman's, 139 ; of influenza, 260 ; pseudo-diphtheria, 138 ; xerosis, 138.

Bacteria, 16 : characters of, 16 ; reproduction of, 16 ; cultivation of, 19 ; aerobic and anaerobic, 21 ; staining of, 22 ; chemical products of, 24 ; pathogenic properties of, 26 ; invasion by, 29, 35 ; destruction of, 29.

Bacteriological examination : in anthrax, 348 ; in diphtheria, 140 ; in influenza, 260.

Baths, in the treatment of fever, 13.

Belladonna, rash produced by, 50.

Bilious fever, 283.

Bull-neck, 73, 82.

Burns and scarlet fever, 77.

Camp fever, 264.

Case mortality. See Fatality.

Catarrhal sore-throat, 53.

Chicken-pox. See Varicella.

Clinical history : of anthrax, 342, 344 ; of chicken-pox, 231 ; of diphtheria, 114 ; of enteric fever, 295 ; of erysipelas, 332 ; of influenza, 255 ; of measles, 166 ; of mumps, 249 ; of relapsing fever, 280 ; of rubeola, 184 ; of scarlet fever, 60 ; of small-pox, 190 ; of typhus fever, 267 ; of vaccinia, 222 ; of whooping-cough, 243.

Cocci, 17.

Coma-vigil, 271.

Complications : of chicken-pox, 235 ; of diphtheria, 126 ; of enteric fever, 306 ; of erysipelas, 334 ; of influenza, 258 ; of measles, 173 ; of mumps, 250 ; of relapsing fever, 283 ; of scarlet fever, 79 ; of small-pox, 202 ; of typhus fever, 272 ; of vaccinia, 224 ; of whooping-cough, 243.

Contagion, 15.

Copaiba, rash produced by, 50.

Cow-pox, 218.

Crisis, 5 ; in erysipelas, 334 ; in measles, 171 ; in relapsing fever, 281 ; in scarlet fever, 67 ; in typhus fever, 271.

Croup, 122.

Desquamation : in drug and other rashes, 49 ; in erysipelas, 333 ; in measles, 172 ; in rubeola, 185 ; in scarlet fever, 67.

Diagnosis : of anthrax, 348 ; of chicken-pox, 239 ; of diphtheria, 145 ; of enteric fever, 321 ; of erysipelas, 338 ; of influenza, 254 ; of measles, 176 ; of mumps, 252 ; of relapsing fever, 286 ; of rubeola, 186 ; of scarlet fever, 93 ; of small-pox, 208, 225, 239 ; of typhus fever, 274 ; of whooping-cough, 246.

Diphtheria, 110 : faucial, 114 ; hæmorrhagic, 121 ; of the genital organs, 121 ; nasal, 122 ; laryngeal, 122 ; of wounds, 125 ; prolonged form of, 126 ; in measles, 174 ; in scarlet fever, 79, 86.

Diplococci, 18.

Disinfection, 41.

Dissemination : of anthrax, 340 ; of chicken-pox, 230 ; of diphtheria, 111 ; of enteric fever, 291 ; of erysipelas, 331 ; of influenza, 254 ; of measles, 165 ; of mumps, 249 ; of relapsing fever, 279 ; of rubeola, 184 ; of scarlet fever, 58 ; of small-pox, 188 ; of typhus fever, 266 ; of whooping-cough, 242.

Drug rashes, 49.

Ehrlich's reaction, in enteric fever, 303.

Enteric fever, 290.

Erysipelas, 330: of fauces, 55, 146.

Erythema scarlatiniforme, 52; morbilliforme, 52; fugax, 52; roseola, 52.

Erythemata simulating specific fevers, 49.

Etiology: of anthrax, 340; of chicken-pox, 230; of diphtheria, 110; of enteric fever, 290; of erysipelas, 330; of influenza, 253; of measles, 165; of mumps, 249; of relapsing fever, 279; of rubeola, 180; of scarlet fever, 57; of small-pox, 188; of typhus fever, 265; of whooping-cough, 242.

Famine fever, 279.

Fastigium, 4.

Fatality: of anthrax, 342, 349; of diphtheria, 113, 152; of enteric fever, 294, 324; of measles, 177; of relapsing fever, 287; of scarlet fever, 57, 59; of small-pox, 211, 228; of typhus fever, 266, 276.

Fever, 1; symptoms of, 4; theory of, 7; causation of, 11; treatment of, 11.

Fever: camp, 264; bilious, 283; enteric, 290; famine, 279; gaol, 264; puerperal, 75, 330; relapsing, 279; scarlet, 57; splenic, 340, 347; spotted, 264; typhoid, 290; typhus, 265; variolous, 201.

Fevers, concurrence of, 34.

Fomites, 15, 41.

Gangrene of fauces (idiopathic), 148.

Gaol fever, 264.

Geographical distribution: of anthrax, 341; of diphtheria, 110; of enteric fever, 290; of erysipelas, 331; of influenza, 254; of measles, 165; of mumps, 249; of relapsing fever, 279; of rubeola, 183; of scarlet fever, 57; of small-pox, 188; of typhus fever, 265; of whooping-cough, 242.

German measles. See Rubeola.

Gram's method of staining bacteria, 23.

Grisolle sign, in small-pox, 210.

Hæmorrhage, intestinal, in enteric fever, 306.

Heat of body, 1; production and loss of, 1; regulation of, 3. See also Fever.

Herpes of palate, etc., 54.

Hoffman's bacillus, 139.

Horn-pox, 202.

Hyperpyrexia, 10.

Ice, in treatment of fever, 13.

Immunity, 28: natural, 28; acquired, 30. See also Protection.

Imperial drink, 106.

Incubation period: of internal anthrax, 344; of chicken-pox, 231; of diphtheria, 114; of enteric fever, 295; of erysipelas, 332; of influenza, 255; of measles, 166; of mumps, 249; of relapsing fever, 280; of rubeola, 184; of scarlet fever, 60; of small-pox, 205; of typhus fever, 273; of whooping-cough, 246. See also Appendix III.

Infection, 15.

Infectivity, Period of: in chicken-pox, 231; in diphtheria, 133; in erysipelas, 336; in influenza, 259; in measles, 175; in mumps, 251; in rubeola, 184; in scarlet fever, 89; in small-pox, 205; in typhus fever, 273; in whooping-cough, 246. See also Appendix III.

Influenza, 253; in animals, 261; febrile type of, 255; bronchitic type of, 256; catarrhal type of, 256; gastro-intestinal type of, 257; cerebral type of, 257.

Initial rashes. See under Rashes.

Inoculation of small-pox, 218.

Insanity, post-febrile: in enteric fever, 309; in influenza, 258; in scarlet fever, 89; in typhus fever, 273.

Intubation, 157.

Isolation, 39. See also Infectivity, Period of; and Appendix III.

Laryngitis: in enteric fever, 309; in erysipelas, 334; in diphtheria, 122; in measles, 173; in scarlet fever, 80; in small-pox, 203.

Laryngitis, membranous, 123.

Leucocytosis, in fever, 7.

Lysis, 5.

Malignant pustule, 340, 342.

Measles, 165. See also Morbilli.

Measles, German, 180.

Membranous inflammation: in acute tonsillitis, 54; in scarlet fever, 79; in measles, 174.

Membranous laryngitis, 123.

Metropolitan Asylums Board, 38; regulations of, *re* removal of patients, Appendix II.

Micro-organisms, 16.

Milk epidemics: of scarlet fever, 58; of diphtheria, 111; of enteric fever, 293.

Morbid anatomy: of anthrax, 345; of chicken-pox, 238; of diphtheria, 134; of enteric fever, 313; of erysipelas, 336; of influenza, 259; of measles, 175; of mumps, 251; of relapsing fever, 284; of scarlet fever, 90; of small-pox, 205; of typhus fever, 273; of whooping-cough, 246.

- Morbilli, 165 : sine catarrho, 172 ; sine morbillis, 173.
Morphia, rash produced by, 51.
Mortality : of anthrax, 342 ; of diphtheria, 110 ; of enteric fever, 290 ;
of influenza, 263 ; of measles, 165, 177 ; of mumps, 252 ; of
scarlet fever, 57 ; of small-pox, 189 ; of whooping-cough, 242,
246.
Mulberry rash, in typhus fever, 270.
Mumps, 249.
- Nephritis : in chicken-pox, 235 ; in diphtheria, 119 ; in enteric fever, 309 ;
in erysipelas, 334 ; in scarlet fever, 82 ; in small-pox, 203 ; in
typhus fever, 273.
Normal unit, 161.
Notifiable diseases, 38.
Notification of Infectious Diseases Act, 37.
- Ophthalmia : in measles, 174 ; in relapsing fever, 284 ; in small-pox, 203.
Orchitis, in mumps, 250.
Otitis : in diphtheria, 132 ; in measles, 174 ; in scarlet fever, 81 ; in
small-pox, 203.
- Paralysis, diphtherial, 126.
Parasitic bacteria, 26.
Parotitis, specific : see Mumps ; in enteric fever, 309.
Pathology : of anthrax, 347 ; of chicken-pox, 238 ; of diphtheria, 142 ;
of enteric fever, 319 ; of erysipelas, 336 ; of influenza, 260 ; of
measles, 175 ; of mumps, 251 ; of relapsing fever, 286 ; of scarlet
fever, 92 ; of small-pox, 205 ; of vaccinia, 225 ; of whooping-
cough, 246.
Perforation : of palate in scarlet fever, 72 ; of intestine in enteric
fever, 307.
Periostitis, in enteric fever, 309.
Peritonitis, in enteric fever, 307.
Pertussis. See Whooping-cough.
Phagocytes, 29.
Potassic bromide, rash produced by, 52.
Potassic iodide, rash produced by, 51, 52.
Prodromal rashes. See Rashes, initial.
Prognosis : in anthrax, 349 ; in chicken-pox, 241 ; in diphtheria, 152 ;
in enteric fever, 324 ; in erysipelas, 338 ; in influenza, 263 ; in
measles, 177 ; in mumps, 252 ; in relapsing fever, 287 ; in rubeola,
187 ; in scarlet fever, 97 ; in small-pox, 211 ; in typhus fever, 276 ;
in whooping-cough, 246.
Prophylactic treatment of infectious diseases, 37. See also Infectivity.

Protection afforded by: chicken-pox, 238; diphtheria, 133; enteric fever, 312; erysipelas, 336; influenza, 259; measles, 175; mumps, 251; relapsing fever, 284; rubeola, 186; scarlet fever, 89; small-pox, 205; typhus fever, 273; vaccination, 226; whooping-cough, 246. See also Appendix III.

Protein, 24.

Protozoa, 16, 285.

Pseudo-diphtheria bacillus, 138.

Puerperal fever, 75, 330; and scarlet fever, 75.

Pyrexia, 4. See also Fever.

Pyrexia, treatment of, 11.

Quarantine, Appendix III.

Quinine, rash produced by, 51.

Quinsy, 54.

Rashes, initial: in chicken-pox, 231; in enteric fever, 299; in measles 167; in small-pox, 190.

Rashes, secondary: in diphtheria, 133; in scarlet fever, 73, 87; after vaccination, 224.

Rashes, simulating specific fevers, 49.

Relapse: in internal anthrax, 345; in chicken-pox, 238; in diphtheria, 133; in enteric fever, 312; in erysipelas, 336; in influenza, 259; in measles, 175; in relapsing fever, 282; in rubeola, 186; in scarlet fever, 89; in typhus fever, 273; in whooping-cough, 246. See also Appendix III.

Relapsing fever, 279.

Revaccination, 223, 227.

Rheumatism (acute): rash in, 52; in relapsing fever, 284; in scarlet fever, 80.

Roetheln. See Rubeola.

Roseola, 52.

Roseola vaccinia, 225.

Rubeola, 180.

Saprophytes, 26.

Sarcinæ, 18.

Scarlatina. See Scarlet fever.

Scarlet fever, 57; simplex, 68; maligna, 69; hæmorrhagic, 70; anginosa, 71; typhoid form of, 75; puerperal, 75; surgical, 77; scarlet fever and burns, 77.

Seasonal prevalence: of chicken-pox, 230; of diphtheria, 111; of enteric fever, 290; of erysipelas, 331; of influenza, 253; of measles, 165; of mumps, 249; of relapsing fever, 280; of rubeola, 183; of scarlet fever, 57; of small-pox, 189; of typhus fever, 265; of whooping-cough, 242.

- Second attacks : of chicken-pox, 238 ; of diphtheria, 133 ; of enteric fever, 313 ; of erysipelas, 336 ; of influenza, 259 ; of measles, 175 ; of mumps, 251 ; of relapsing fever, 284 ; of rubeola, 186 ; of scarlet fever, 89 ; of small-pox, 205 ; of typhus fever, 273 ; of whooping-cough, 246. See also Appendix III.
- Secondary rashes. See Rashes.
- Septic diseases, 27 : immunity in, 31 ; recovery from, 33.
- Septicæmia, rash in, 52.
- Serum, for diphtheria and tetanus, rash produced by, 51, 160 ; Appendix IV.
- Serum treatment of diphtheria, 160, and Appendix IV.
- Sex-incidence : of diphtheria, 114 ; of enteric fever, 294 ; of mumps, 249 ; of relapsing fever, 280 ; of rubeola, 184 ; of scarlet fever, 59.
- Small-pox, 188 ; discrete, 197 ; semi-confluent or coherent, 197 ; confluent, 199 ; modified, 200 ; hæmorrhagic, 203.
- Sore-throat, various forms of, 53.
- Spirillum of relapsing fever, 285.
- Spirochæta Obermeieri, 285.
- Splenic fever, 340.
- Spores, 17.
- Spotted fever, 264.
- Staphylococci, 18.
- Statistics. See Tables.
- Stomatitis, in scarlet fever, 72, 87.
- Streptococci, 18 : in diphtheria, 144 ; in erysipelas, 336 ; in scarlet fever, 92.
- Surgical scarlet fever, 77.
- Symptoms of various specific diseases. See Clinical History.
- Syphilis, sore throat in, 55.
- Tables, statistical : of anthrax, 342 ; of diphtheria, 113, 162, Appendix IV. ; of enteric fever, 294 ; of relapsing fever, 280 ; of scarlet fever, 59, 60 ; of typhus fever, 266.
- Temperature. See Fever, and Heat.
- Thrush, 147.
- Tonsillitis, various forms of, 54.
- Toxic diseases, 27 : immunity in, 31 ; recovery from, 33.
- Toxines, 24, 33.
- Tracheotomy, 157.
- Treatment : of anthrax, 350 ; of chicken-pox, 241 ; of diphtheria, 154 ; of enteric fever, 326 ; of erysipelas, 338 ; of influenza, 263 ; of measles, 177 ; of mumps, 252 ; of pyrexia, 11 ; of relapsing fever, 288 ; of rubeola, 187 ; of scarlet fever, 101 ; of small-pox, 213 ; of typhus fever, 276 ; of whooping-cough, 247.

- Treatment, antitoxine, 160, and Appendix IV.
Tuberculin, rash produced by, 51.
Tuberculosis: after chicken-pox, 241 ; after measles, 175 ; after whooping-cough, 245.
Tuberculous ulceration of fauces, 55.
Typhoid bacillus, 317.
Typhoid fever. See Enteric fever.
Typhus fever, 265 ; typhus siderans, 272.
- Ulcerated sore-throat, 53.
Unit, normal, 161.
Uræmia, rash in, 52.
Urine, suppression of. See Anuria.
- Vaccination, 212, 219.
Vaccinia : see Vaccination ; generalised, 225.
Varicella, 230 : gangrenosa, 235 ; bullosa, 238 ; modified, 238.
Varieties: of chicken-pox, 237 ; of diphtheria, 119 ; of enteric fever, 304 ; of influenza, 255 ; of measles, 172 ; of relapsing fever, 282 ; of scarlet fever, 68 ; of small-pox, 197 ; of typhus fever, 272 ; of whooping-cough, 244.
Variola : see Small-pox ; cornea, 202 ; nigra, 203 ; sine variolis, 201 ; verrucosa, 202.
Variolation, 218.
Varioloid, 200.
Variolous fever, 201.
Vibrios, 17.
- Wart-pox, 202.
Water, treatment of pyrexia by, 12.
Wet-pack, 12.
Whooping-cough, 175, 242.
Wool-sorter's disease, 340.
Wounds, diphtheria of, 125.
- Xerosis bacillus, 138.



January, 1897.

CATALOGUE OF WORKS

PUBLISHED BY

H. K. LEWIS

136 GOWER STREET, LONDON, W.C.

Established 1844.

A. C. ABBOTT, M.D.

First Assistant, Laboratory of Hygiene, University of Pennsylvania.

THE PRINCIPLES OF BACTERIOLOGY: A Practical
Manual for Students and Physicians. Third Edition, with 98 Illustrations, 17 being coloured, post 8vo, 12s. 6d. *nett*.

H. ALDERSMITH, M.B. LOND., F.R.C.S.

Resident Medical Officer, Christ's Hospital, London.

RINGWORM: Its Diagnosis and Treatment. Fourth Edition, enlarged, with Illustrations, fcap. 8vo. [In preparation.]

HARRISON ALLEN, M.D.

Consulting Physician to Rush Hospital for Consumption.

A HANDBOOK OF LOCAL THERAPEUTICS. General Surgery by R. H. HARTE, M.D., Surgeon to the Episcopal and St. Mary's Hospitals; Diseases of the Skin by A. VAN HARLINGEN, M.D., Professor of Diseases of the Skin in the Philadelphia Polyclinic; Diseases of the Ear and Air Passages by H. ALLEN, M.D.; Diseases of the Eye by G. C. HARLAN, M.D., Surgeon to Wills Eye Hospital. Edited by H. ALLEN, M.D. Large 8vo, 14s. *nett*.

JAMES ANDERSON, M.D., F.R.C.P.

Late Assistant Physician to the London Hospital, &c.

NOTES ON MEDICAL NURSING from the Lectures given to the Probationers at the London Hospital. Edited by E. F. LAMPORT, Associate of the Sanitary Institute. With an Introductory Biographical Notice by the late SIR ANDREW CLARK, BART. Third Edition, crown 8vo, 2s. 6d. [Just ready.]

E. CRESSWELL BABER, M.B. LOND.

Surgeon to the Brighton and Sussex Throat and Ear Hospital.

A GUIDE TO THE EXAMINATION OF THE NOSE, WITH REMARKS ON THE DIAGNOSIS OF DISEASES OF THE NASAL CAVITIES. With Illustrations, small 8vo, 5s. 6d.

JAMES B. BALL, M.D. LOND., M.R.C.P.

Physician to the Department for Diseases of the Throat and Nose, and Senior Assistant Physician, West London Hospital.

INTUBATION OF THE LARYNX. With Illustrations, demy 8vo, 2s. 6d.

G. GRANVILLE BANTOCK, M.D., F.R.C.S. EDIN.

Surgeon to the Samaritan Free Hospital for Women and Children.

I.

ON THE USE AND ABUSE OF PESSARIES. Second Edition, with Illustrations, 8vo, 5s.

II.

ON THE TREATMENT OF RUPTURE OF THE FEMALE PERINEUM IMMEDIATE AND REMOTE. Second Edition, with Illustrations, 8vo, 3s. 6d.

III.

A PLEA FOR EARLY OVARIOTOMY. Demy 8vo, 2s

FANCOURT BARNES, M.D., M.R.C.P.

Physician to the Chelsea Hospital for Women; Obstetric Physician to the Great Northern Hospital, &c.

A GERMAN-ENGLISH DICTIONARY OF WORDS AND TERMS USED IN MEDICINE AND ITS COGNATE SCIENCES. Square 12mo, Roxburgh binding, 9s.

JAMES BARR, M.D.

Physician to the Northern Hospital, Liverpool; Medical Officer of Her Majesty's Prison, Kirkdale, &c.

THE TREATMENT OF TYPHOID FEVER, and reports of fifty-five consecutive cases with only one death. With Introduction by W. T. GAIRDNER, M.D., LL.D., Professor of Medicine in the University of Glasgow. With Illustrations, demy 8vo, 6s.

ASHLEY W. BARRETT, M.B. LOND., M.R.C.S., L.D.S.E.

Dental Surgeon to, and Lecturer on Dental Surgery in the Medical School of, the London Hospital; Examiner in Dental Surgery to the Royal College of Surgeons, England.

DENTAL SURGERY FOR MEDICAL PRACTITIONERS AND STUDENTS OF MEDICINE. Third Edition, with Illustrations, cr. 8vo, 3s. 6d.

[LEWIS'S PRACTICAL SERIES.]

[Just ready.]

ROBERTS BARTHOLOW, M.A., M.D., LL.D.

Professor Emeritus of Materia Medica, General Therapeutics, and Hygiene in the Jefferson Medical College of Philadelphia, &c., &c.

I.

A PRACTICAL TREATISE ON MATERIA MEDICA AND THERAPEUTICS. Ninth Edition, large 8vo, 21s. [Now ready.]

II.

A TREATISE ON THE PRACTICE OF MEDICINE, FOR THE USE OF STUDENTS AND PRACTITIONERS. Fifth Edition, with Illustrations, large 8vo, 21s.

H. CHARLTON BASTIAN, M.A., M.D., F.R.S., F.R.C.P.

Professor of the Principles and Practice of Medicine in University College, London; Physician to University College Hospital, &c.

I.

PARALYSES: CEREBRAL, BULBAR, AND SPINAL. A MANUAL OF DIAGNOSIS FOR STUDENTS AND PRACTITIONERS. With numerous Illustrations, 8vo, 12s. 6d.

II.

VARIOUS FORMS OF HYSTERICAL OR FUNCTIONAL PARALYSIS. Demy 8vo, 7s. 6d.

W. M. BEAUMONT.

Surgeon to the Bath Eye Infirmary.

THE SHADOW-TEST IN THE DIAGNOSIS AND ESTIMATION OF AMETROPIA. Post 8vo, 2s. 6d.

F. E. BEDDARD, M.A., F.R.S.

[See Cambridge Natural Science Manuals, page 5.]

E. H. BENNETT, M.D., F.R.C.S.I.

Professor of Surgery, University of Dublin,

AND

D. J. CUNNINGHAM, M.D., F.R.C.S.I.

Professor of Anatomy and Chirurgery, University of Dublin.

THE SECTIONAL ANATOMY OF CONGENITAL CÆCAL HERNIA. With coloured plates, sm. folio, 5s. 6d.

A. HUGHES BENNETT, M.D., M.R.C.P.

Physician to the Hospital for Epilepsy and Paralysis, Regent's Park, &c.

A PRACTICAL TREATISE ON ELECTRO-DIAGNOSIS IN DISEASES OF THE NERVOUS-SYSTEM. With Illustrations, 8vo, 8s. 6d.

HORATIO R. BIGELOW, M.D.

Permanent Member of the American Medical Association, &c.

PLAIN TALKS ON ELECTRICITY AND BATTERIES, WITH THERAPEUTIC INDEX, FOR GENERAL PRACTITIONERS AND STUDENTS OF MEDICINE. With Illustrations, crown 8vo, 4s. 6d.

DRS. BOURNEVILLE AND BRICON.

MANUAL OF HYPODERMIC MEDICATION.

Translated from the Second Edition, and Edited, with Therapeutic Index of Diseases, by ANDREW S. CURRIE, M.D. Edin., &c. With Illustrations, crown 8vo, 3s. 6d.

ROBERT BOXALL, M.D. CANTAB., M.R.C.P. LOND.

Assistant Obstetric Physician at the Middlesex Hospital, &c.

ANTISEPTICS IN MIDWIFERY. 8vo. 1s.

RUBERT BOYCE, M.B., M.R.C.S.

Professor of Pathology in University College, Liverpool.

A TEXT-BOOK OF MORBID HISTOLOGY for Students and Practitioners. With 130 coloured Illustrations, royal 8vo, 31s. 6d.

GURDON BUCK, M.D.

CONTRIBUTIONS TO REPARATIVE SURGERY:

Showing its Application to the Treatment of Deformities, produced by Destructive Disease or Injury; Congenital Defects from Arrest or Excess of Development; and Cicatricial Contractions from Burns. Large 8vo, 9s.

MARY BULLAR & J. F. BULLAR, M.B. CANTAB., F.R.C.S.

RECEIPTS FOR FLUID FOODS. 16mo, 1s.

CHARLES H. BURNETT, A.M., M.D.

Emeritus Professor of Otolaryngology in the Philadelphia Polyclinic; Clinical Professor of Otolaryngology in the Woman's Medical College of Pennsylvania; Aural Surgeon to the Presbyterian Hospital, Philadelphia.

SYSTEM OF DISEASES OF THE EAR, NOSE AND

THROAT. By 45 Eminent American, British, Canadian and Spanish Authors. Edited by CHARLES H. BURNETT. With Illustrations, in two imperial 8vo vols., half morocco, 48s. *nett*.

STEPHEN SMITH BURT, M.D.

Professor of Clinical Medicine and Physical Diagnosis in the New York Post-graduate School and Hospital.

EXPLORATION OF THE CHEST IN HEALTH AND DISEASE. With Illustrations, crown 8vo, 6s.

DUDLEY W. BUXTON, M.D., B.S., M.R.C.P.

Administrator of Anæsthetics and Lecturer in University College Hospital, the National Hospital for Paralysis and Epilepsy, Queen's Square, and the Dental Hospital of London.

ANÆSTHETICS THEIR USES AND ADMINISTRATION. Second Edition, with Illustrations, crown 8vo, 5s.

[LEWIS'S PRACTICAL SERIES.]

HARRY CAMPBELL, M.D., B.S. LOND., M.R.C.P.

Physician to the North-West London Hospital.

THE CAUSATION OF DISEASE: An exposition of the ultimate factors which induce it. Demy 8vo, 12s. 6d.

FLUSHING AND MORBID BLUSHING: THEIR PATHOLOGY AND TREATMENT. With plates and wood engravings, royal 8vo, 10s. 6d.

DIFFERENCES IN THE NERVOUS ORGANISATION OF MAN AND WOMAN, PHYSIOLOGICAL AND PATHOLOGICAL. Royal 8vo, 15s.

HEADACHE AND OTHER MORBID CEPHALIC SENSATIONS. Royal 8vo, 12s. 6d.

CAMBRIDGE NATURAL SCIENCE MANUALS.

BIOLOGICAL SERIES.

(General Editor : A. E. SHIPLEY, M.A., Fellow and Tutor of Christ's College).

ELEMENTARY PALÆONTOLOGY—INVERTEBRATE.

By HENRY WOODS, B.A., F.G.S. Crown 8vo, 6s.

PRACTICAL PHYSIOLOGY OF PLANTS.

By F. DARWIN, M.A., F.R.S. and E. H. ACTON, M.A. Second Edition, crown 8vo, 6s.

PRACTICAL MORBID ANATOMY.

By H. D. ROLLESTON, M.D., F.R.C.P., Fellow of St. John's College, Cambridge; Assistant Physician and Lecturer on Pathology, St. George's Hospital, London; and A. A. KANTHACK, M.D., M.R.C.P., Lecturer on Pathology, St. Bartholomew's Hospital, London. Crown 8vo, 6s.

ZOOGEOGRAPHY.

By F. E. BEDDARD, M.A., F.R.S. Crown 8vo, 6s.

ELEMENTS OF BOTANY.

By F. DARWIN, M.A., F.R.S. Second Edition, crown 8vo, 6s.

FOSSIL PLANTS. A Manual for Students of Botany and Geology.

By A. C. SEWARD, M.A., F.G.S.

[In preparation.]

PHYSICAL SERIES.

(General Editor : R. T. GLAZEBROOK, M.A., F.R.S., Fellow of Trinity College; Assistant Director of the Cavendish Laboratory).

HEAT AND LIGHT.

By R. T. GLAZEBROOK. Crown 8vo, 5s. (The two parts are also published separately). HEAT, 3s. LIGHT, 3s.

MECHANICS AND HYDROSTATICS.

By the same Author. Crown 8vo, 8s. 6d. Part I.—DYNAMICS, 4s. Part II.—STATICS, 3s. Part III.—HYDROSTATICS, 3s.

ELECTRICITY AND MAGNETISM.

By the same Author.

[In preparation.]

SOLUTION AND ELECTROLYSIS.

By W. C. D. WHETHAM, M.A., Fellow of Trinity College. Crown 8vo, 7s. 6d.

ELEMENTS OF PETROLOGY.

By A. HARKER, M.A., F.G.S., Fellow of St. John's College; University Demonstrator of Petrology. Crown 8vo, 7s. 6d.

R. E. CARRINGTON, M.D., F.R.C.P.

Late Assistant Physician and Senior Demonstrator of Morbid Anatomy at Guy's Hospital.

NOTES ON PATHOLOGY. With an Introductory Chapter by J. F. GOODHART, M.D. (ABERD.), F.R.C.P., Physician to Guy's Hospital, Edited by H. EVELYN CROOK, M.D. LOND., F.R.C.S. ENG., and GUY MACKESON, L.R.C.P., M.R.C.S. Crown 8vo, 3s. 6d.

ALFRED H. CARTER, M.D. LOND.

Fellow of the Royal College of Physicians; Senior Physician to the Queen's Hospital, Birmingham; Professor of Therapeutics in Mason College, Birmingham.

ELEMENTS OF PRACTICAL MEDICINE.

Seventh Edition, crown 8vo, 10s.

[Now ready.]

F. H. CHAMPNEYS, M.A., M.D. OXON., F.R.C.P.

Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; Examiner in Obstetric Medicine in the University of Oxford, &c.

I.

LECTURES ON PAINFUL MENSTRUATION. THE HARVEIAN LECTURES, 1890. Roy. 8vo, 7s. 6d.

II.

EXPERIMENTAL RESEARCHES IN ARTIFICIAL RESPIRATION IN STILLBORN CHILDREN, AND ALLIED SUBJECTS. Crown 8vo, 3s. 6d.

W. BRUCE CLARKE, M.A., M.B. OXON., F.R.C.S.

Assistant Surgeon to, and Senior Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; Surgeon to the West London Hospital, &c.

THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE KIDNEY AMENABLE TO DIRECT SURGICAL INTERFERENCE. With Illustrations, demy 8vo, 7s. 6d.

JOHN COCKLE, M.A., M.D.

Physician to the Royal Free Hospital.

ON INTRA-THORACIC CANCER. 8vo, 4s. 6d.

ALEXANDER COLLIE, M.D. ABERD., M.R.C.P. LOND.

Secretary of the Epidemiological Society for Germany and Russia, &c.

ON FEVERS: THEIR HISTORY, ETIOLOGY, DIAGNOSIS, PROGNOSIS, AND TREATMENT. Illustrated with Coloured Plates, crown 8vo, 8s. 6d. [LEWIS'S PRACTICAL SERIES.]

M. P. MAYO COLLIER, M.B., M.S. LOND., F.R.C.S. ENG.

Professor of Comparative Anatomy and Physiology at the Royal College of Surgeons, England, &c.

THE PHYSIOLOGY OF THE VASCULAR SYSTEM. Illustrations, 8vo, 3s. 6d.

E. TREACHER COLLINS, F.R.C.S.

Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Hunterian Professor, Royal College of Surgeons, England, 1893-94.

RESEARCHES INTO THE ANATOMY AND PATHOLOGY OF THE EYE. With 10 Plates and 28 Figures in the Text, demy 8vo, 6s. [Now ready.]

WALTER S. COLMAN, M.D., M.R.C.P. LOND.

Assistant Physician to the National Hospital for the Paralysed and Epileptic, &c.

SECTION CUTTING AND STAINING: A Practical
Introduction to Histological Methods for Students and Practitioners.
Second Edition, with Illustrations, crown 8vo, 3s. 6d. [Now ready.]

W. H. CORFIELD, M.A., M.D. OXON., F.R.C.P. LOND.

Professor of Hygiene and Public Health in University College, London; Medical Officer of Health for St. George's, Hanover Square, &c.

DWELLING HOUSES: their Sanitary Construction and
Arrangements. Fourth Edition, with Illustrations, crown 8vo, 3s. 6d.
[Just ready.]

II.

DISEASE AND DEFECTIVE HOUSE SANITATION:
being Two Lectures delivered before the Harveian Society of London.
With Illustrations, crown 8vo, 2s. [Just published.]

J. LEONARD CORNING, M.A., M.D.

Consultant in Nervous Diseases to St. Francis Hospital.

A PRACTICAL TREATISE ON HEADACHE, NEURALGIA, SLEEP AND ITS DERANGEMENTS, AND SPINAL IRRITATION. With an Appendix—Eye Strain, a Cause of Headache
By DAVID WEBSTER, M.D. Second Edition, demy 8vo, 7s. 6d.

SIDNEY COUPLAND, M.D., F.R.C.P.

Physician to the Middlesex Hospital, and Lecturer on Practical Medicine in the Medical School; late Examiner in Medicine at the Examining Board for England.

NOTES ON THE CLINICAL EXAMINATION OF THE
BLOOD AND EXCRETA. Third Edition, 12mo, 1s. 6d.

CHARLES CREIGHTON, M.A., M.D.

Formerly Demonstrator of Anatomy in the University of Cambridge.

A HISTORY OF EPIDEMICS IN BRITAIN, Vol. I.
FROM A.D. 664 TO THE EXTINCTION OF THE PLAGUE.
Demy 8vo, 18s. Vol. II. FROM THE EXTINCTION OF THE
PLAGUE TO THE PRESENT TIME. Demy 8vo, 20s.

II.

ILLUSTRATIONS OF UNCONSCIOUS MEMORY IN
DISEASE, including a Theory of Alternatives. Post 8vo, 6s.

III.

CONTRIBUTIONS TO THE PHYSIOLOGY AND
PATHOLOGY OF THE BREAST AND LYMPHATIC GLANDS.
New Edition with additional chapter, with wood-cuts and plate, 8vo, 9s.

IV.

BOVINE TUBERCULOSIS IN MAN: An Account of the
Pathology of Suspected Cases. With Chromo-lithographs and other
Illustrations, 8vo, 8s. 6d.

H. RADCLIFFE CROCKER, M.D. LOND., B.S., F.R.C.P.

Physician for Diseases of the Skin in University College Hospital, &c.

DISEASES OF THE SKIN; THEIR DESCRIPTION,
PATHOLOGY, DIAGNOSIS, AND TREATMENT. Second Edition,
with 92 Illustrations, 8vo, 24s.

EDGAR M. CROOKSHANK, M.B. LOND.

Professor of Comparative Pathology and Bacteriology, and Fellow of, King's College, London

A TEXTBOOK OF BACTERIOLOGY: Including the Etiology and Prevention of Infective Diseases, and a short account of Yeasts and Moulds, Hæmatozoa, and Psorosperms. Illustrated with 22 Coloured Plates, and 273 Illustrations in the Text. Fourth Edition, medium 8vo, 21s. *nett.* [Just published.]

II.

HISTORY AND PATHOLOGY OF VACCINATION. Vol. I., A Critical Inquiry. Vol. II., Selected Essays, (Edited) including works by Jenner, Pearson, Woodville, Henry Jenner, Loy, Rogers, Birch, Bousquet, Estlin, Ceely, Badcock, Auzias-Turenne, Dubreuilh and Layet. Two volumes, illustrated with 22 coloured plates. Royal 8vo, 20s. *nett.*

F. DARWIN, M.A., F.R.S.

[See Cambridge Natural Science Manuals, page 5.]

HERBERT DAVIES, M.D., F.R.C.P.

Late Consulting Physician to the London Hospital.

THE MECHANISM OF THE CIRCULATION OF THE BLOOD THROUGH ORGANICALLY DISEASED HEARTS. Edited by ARTHUR TEMPLER DAVIES, B.A. (Nat. Science Honours), M.D. Cantab., M.R.C.P.; Physician to the Royal Hospital for Diseases of the Chest. Crown 8vo, 3s. 6d.

HENRY DAVIS, M.R.C.S.

Teacher and Administrator of Anæsthetics at St. Mary's Hospital, and Anæsthetist to the Dental Hospital of London.

GUIDE TO THE ADMINISTRATION OF ANÆSTHETICS. Second Edition, fcap. 8vo, 2s. 6d.

F. A. DIXEY, M.A., D.M.

Fellow of Wadham College, Oxford.

EPIDEMIC INFLUENZA: A Study in Comparative Statistics. With Diagrams and Tables. 8vo, 7s. 6d.

HORACE DOBELL, M.D.

Consulting Physician to the Royal Hospital for Diseases of the Chest, &c.

ON DIET AND REGIMEN IN SICKNESS AND Health, and on the Interdependence and Prevention of Diseases and the Diminution of their Fatality. Seventh Edition, 8vo, 5s. *nett.*

ROBERT W. DOYNE, F.R.C.S.

Surgeon to the Oxford Eye Hospital; Ophthalmic Surgeon to St. John's Hospital, Cowley, and to the Bourton on Water Cottage Hospital.

NOTES ON THE MORE COMMON DISEASES OF THE EYE. With Test Types, crown 8vo, 2s. [Now ready.]

DR. A. DUHRSEN.

Privat Dozent in Midwifery and Gynæcology in the University of Berlin.

I.

A MANUAL OF GYNÆCOLOGICAL PRACTICE FOR STUDENTS AND PRACTITIONERS. Translated and Edited from the Fourth German edition, by JOHN W. TAYLOR, F.R.C.S., Surgeon to the Birmingham and Midland Hospital for Women, &c.; and FREDERICK EDGE, M.D. LOND., M.R.C.P., F.R.C.S., Surgeon to the Wolverhampton and District Hospital for Women. With 120 Illustration, crown 8vo, 6s. [Now ready.]

II.

A MANUAL OF OBSTETRIC PRACTICE FOR STUDENTS AND PRACTITIONERS. Translated from the Sixth German Edition, with Illustrations, crown 8vo. [In the press.]

JOHN EAGLE.

Member of the Pharmaceutical Society.

A NOTE-BOOK OF SOLUBILITIES. Arranged chiefly for the use of Prescribers and Dispensers. 12mo, 2s. 6d.

ARTHUR W. EDIS, M.D. LOND., F.R.C.P.

Senior Physician to the Chelsea Hospital for Women; Late Obstetric Physician to the Middlesex Hospital.

STERILITY IN WOMEN: including its Causation and Treatment. With 33 Illustrations, demy 8vo, 6s.

W. ELDER, M.D., F.R.C.P. EDIN.

Physician to Leith Hospital.

APHASIA AND THE CEREBRAL SPEECH MECHANISM. With Illustrations, demy 8vo. [In preparation.]

ALEXANDER S. FAULKNER.

Surgeon-Major, Indian Medical Service.

A GUIDE TO THE PUBLIC MEDICAL SERVICES. Compiled from Official Sources. 8vo, 2s.

W. SOLTAU FENWICK, M.D., B.S. LOND.

Member of the Royal College of Physicians; Assistant Physician to the Evelina Hospital for Sick Children, &c.

I.

THE DYSPEPSIA OF PHTHISIS: Its Varieties and Treatment, including a Description of Certain Forms of Dyspepsia associated with the Tubercular Diathesis. Demy 8vo, 6s.

II.

DISORDERS OF DIGESTION IN INFANCY AND CHILDHOOD. With Illustrations, demy 8vo. [In preparation.]

DR. FERBER.

MODEL DIAGRAM OF THE ORGANS IN THE THORAX AND UPPER PART OF THE ABDOMEN. With Letter-press Description. In 4to, coloured, 5s. nett.

J. MAGEE FINNY, M.D. DUBL.

King's Professor of Practice of Medicine in School of Physic, Ireland, &c.

NOTES ON THE PHYSICAL DIAGNOSIS OF LUNG DISEASES. 32mo, 1s. 6d.

AUSTIN FLINT, M.D., LL.D.

Professor of Physiology and Physiological Anatomy in the Bellevue Hospital Medical College, New York; Visiting Physician to the Bellevue Hospital, &c.

A TEXT-BOOK OF HUMAN PHYSIOLOGY. Fourth Edition, Illustrated by plates, and 316 wood engravings, large 8vo, 25s.

W. H. RUSSELL FORSBROOK, M.D. LOND., M.R.C.S.

Consulting Medical Officer to the Government of the Cape of Good Hope; formerly Surgical Registrar to Westminster Hospital.

A DISSERTATION ON OSTEO-ARTHRITIS. Demy 8vo, 5s.

J. MILNER FOTHERGILL, M.D., M.R.C.P.

Late Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, &c.

I.

INDIGESTION AND BILIOUSNESS. Second Edition, post 8vo, 7s. 6d.

II.

GOUT IN ITS PROTEAN ASPECTS. Post 8vo, 7s. 6d.

III.

THE TOWN DWELLER: His Needs and His Wants.
With an Introduction by SIR B. W. RICHARDSON, M.D., LL.D., F.R.S.
Post 8vo, 3s. 6d.

PROFESSOR E. FUCHS.

Professor of Ophthalmology in the University of Vienna.

TEXTBOOK OF OPHTHALMOLOGY.

Translated from the German by A. DUANE, M.D., Assistant Surgeon,
Ophthalmic and Aural Institute, New York. Second Edition, with
190 Illustrations, large octavo, 21s.

PROFESSOR DR. PAUL FÜRBRINGER.

Director of the Friedrichshain Hospital, Berlin, &c.

**TEXT-BOOK OF DISEASES OF THE KIDNEYS AND
GENITO-URINARY ORGANS.** Translated by W. H. GILBERT,
M.D., Physician in Baden-Baden, &c. Vol. I., demy 8vo, 7s. 6d.

SIR DOUGLAS GALTON.

*Late Royal Engineers, K.C.B., Hon. D.C.L., LL.D., F.R.S., Assoc. Inst. C.E., M.I.Mech.E.,
F.S.A., F.G.S., F.L.S., F.C.S., F.R.G.S., &c.*

HEALTHY HOSPITALS. Observations on some points
connected with Hospital Construction. With Illustrations, 8vo, 10s. 6d.

JOHN HENRY GARRETT, M.D.

*Licentiate in Sanitary Science and Diplomate in Public Health, Universities of Durham
and Cambridge, &c.*

THE ACTION OF WATER ON LEAD; being an inquiry
into the Cause and Mode of the Action and its Prevention. Crown 8vo,
4s. 6d.

ALFRED W. GERRARD, F.C.S.

Examiner to the Pharmaceutical Society, &c.

I.
**ELEMENTS OF MATERIA MEDICA AND PHAR-
MACY.** Crown 8vo, 8s. 6d.

II.

NEW OFFICIAL REMEDIES, B.P., 1890. Supplement
to the above. Crown 8vo, 1s.

HENEAGE GIBBES, M.D.

Lecturer on Physiology in the Medical School of Westminster Hospital, &c.

PRACTICAL HISTOLOGY AND PATHOLOGY.

Third Edition, revised and enlarged, crown 8vo, 6s.

R. T. GLAZEBROOK, M.A., F.R.S.

[See Cambridge Natural Science Manuals, page 5.]

E. W. GOODALL, M.D. LOND.

*Medical Superintendent of the Eastern Hospital of the Metropolitan Asylums Board
Formerly Medical Registrar to Guy's Hospital,*

AND

J. W. WASHBOURN, M.D. LOND.

*Fellow of the Royal College of Physicians; Physician to the London Fever Hospital;
Assistant Physician to Guy's Hospital, and Lecturer in the Medical School.*

A MANUAL OF INFECTIOUS DISEASES. Demy 8vo,
illustrated with Plates, Diagrams, and Charts, 15s. [Now ready.]

JAMES F. GOODHART, M.D. ABERD., F.R.C.P.
Physician to Guy's Hospital, and Consulting Physician to the Evelina Hospital for Sick Children.

ON COMMON NEUROSES: or the Neurotic Element in Disease and its Rational Treatment. Second Edition, crown 8vo, 3s. 6d.

C. A. GORDON, M.D., C.B.
Deputy Inspector General of Hospitals, Army Medical Department.

REMARKS ON ARMY SURGEONS AND THEIR WORKS. Demy 8vo, 5s.

JOHN GORHAM, M.R.C.S.

TOOTH EXTRACTION: a Manual on the proper mode of extracting Teeth. Fourth Edition, fcap. 8vo, 1s. 6d.

GEORGE M. GOULD, A.M., M.D.
Ophthalmic Surgeon to the Philadelphia Hospital, &c.

I.
THE STUDENT'S MEDICAL DICTIONARY: including all the words and phrases generally used in Medicine, with their proper pronunciation and definitions, based on recent medical literature. Tenth Edition, 8vo, 14s. *nett*.

II.
A POCKET MEDICAL DICTIONARY, Giving the Pronunciation and Definition of about 12000 of the Principal Words used in Medicine and the Collateral Sciences. 32mo, 4s. *nett*.

W. R. GOWERS, M.D., F.R.C.P., M.R.C.S.
Physician to University College Hospital, &c.

DIAGRAMS FOR THE RECORD OF PHYSICAL SIGNS.
 In books of 12 sets of figures, 1s.

SURGEON-CAPTAIN A. E. GRANT, M.B.
Indian Medical Service; Professor of Hygiene, Madras Medical College.
THE INDIAN MANUAL OF HYGIENE.
 Vol. I., 8vo, 10s. 6d. *nett*.

LANDON C. GRAY, M.D.
Professor of Nervous and Mental Diseases in the New York Polyclinic; Visiting Physician to St. Mary's Hospital, &c.

A TREATISE ON NERVOUS AND MENTAL DISEASES FOR STUDENTS AND PRACTITIONERS OF MEDICINE. With 168 illustrations, 8vo, 21s.

J. B. GRESSWELL, M.R.C.V.S.
Provincial Veterinary Surgeon to the Royal Agricultural Society.
VETERINARY PHARMACOLOGY AND THERAPEUTICS. With an Index of Diseases and Remedies. Fcap. 8vo, 5s.

A. HILL GRIFFITH, M.D.
Surgeon, Manchester Royal Eye Hospital.
THE DIAGNOSIS OF INTRA-OCULAR GROWTHS.
 With 8 woodcuts, 8vo, 1s. 6d.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. OXON.
Professor of Surgery in the Jefferson Medical College of Philadelphia.
A PRACTICAL TREATISE ON THE DISEASES, INJURIES, AND MALFORMATIONS OF THE URINARY BLADDER, THE PROSTATE GLAND, AND THE URETHRA. Third Edition, revised and edited by S. W. GROSS, A.M., M.D., Surgeon to the Philadelphia Hospital. Illustrated by 170 engravings, 8vo, 18s.

SAMUEL W. GROSS, A.M., M.D.

Surgeon to, and Lecturer on Clinical Surgery in, the Jefferson Medical College Hospital and the Philadelphia Hospital, &c.

- A PRACTICAL TREATISE ON TUMOURS OF THE MAMMARY GLAND:** embracing their Histology, Pathology, Diagnosis, and Treatment. With Illustrations, 8vo, 10s. 6d.

DR. JOSEF GRUBER.

Professor of Otolaryngology in the University of Vienna, etc.

- A TEXT-BOOK OF THE DISEASES OF THE EAR.** Translated from the Second German edition, and Edited, with additions, by EDWARD LAW, M.D., C.M. EDIN., M.R.C.S. ENG., Surgeon to the London Throat Hospital for Diseases of the Throat, Nose and Ear; and COLEMAN JEWELL, M.B. LOND., M.R.C.S. ENG., late Surgeon and Pathologist to the London Throat Hospital. Second English Edition, with 165 Illustrations, and 70 coloured figures on 2 lithographic plates, royal 8vo, 28s.

F. DE HAVILLAND HALL, M.D., F.R.C.P. LOND.

Physician to Out-patients, and in charge of the Throat Department at the Westminster Hospital, &c.

- DISEASES OF THE NOSE AND THROAT.** Crown 8vo, with 2 coloured plates and 59 illustrations, 10s. 6d. [Now ready.]
[LEWIS'S PRACTICAL SERIES.]

WILLIAM A. HAMMOND, M.D.

Professor of Mental and Nervous Diseases in the Medical Department of the University of the City of New York, &c.

- SPIRITUALISM AND ALLIED CAUSES AND CONDITIONS OF NERVOUS DERANGEMENT.** With Illustrations, post 8vo, 8s. 6d.

A. HARKER, M.A., F.G.S.

[See Cambridge Natural Science Manuals, page 5.]

VINCENT DORMER HARRIS, M.D. LOND., F.R.C.P.

Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Examining Physician to the Royal National Hospital for Consumption and Diseases of the Chest, Ventnor, &c.

AND

EDWIN CLIFFORD BEALE, M.A., M.B. CANTAB., F.R.C.P.

Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, and to the Great Northern Central Hospital, &c.

- THE TREATMENT OF PULMONARY CONSUMPTION.** A Practical Manual. Crown 8vo, 10s. 6d. [Now ready.]

[LEWIS'S PRACTICAL SERIES.]

ALEXANDER HARVEY, M.D.

Late Emeritus Professor of Materia Medica in the University of Aberdeen, &c.,

AND

ALEXANDER DYCE DAVIDSON, M.D., F.R.S. EDIN.

Late Regius Professor of Materia Medica in the University of Aberdeen.

- SYLLABUS OF MATERIA MEDICA FOR THE USE OF STUDENTS, TEACHERS AND PRACTITIONERS.** Based on the relative values of articles and preparations in the British Pharmacopœia. Ninth Edition, 32mo, 1s. 6d.

W. S. HEDLEY, M.D.

In charge of the Electro-Therapeutic Department of the London Hospital.

I.
THE HYDRO-ELECTRIC METHODS IN MEDICINE.
Second Edition, with Illustrations, demy 8vo, 4s. 6d. [Now ready.]

II.
CURRENT FROM THE MAIN: The Medical Employment of Electric Lighting Currents. With Illustrations, demy 8vo, 2s. 6d. [Now ready.]

H. HELBING, F.C.S.

MODERN MATERIA MEDICA: FOR MEDICAL MEN, PHARMACISTS, AND STUDENTS. Fourth Edition, 8vo, 8s. nett. [Just published.]

C. HIGGENS, F.R.C.S.

Ophthalmic Surgeon to Guy's Hospital; Lecturer on Ophthalmology at Guy's Hospital Medical School

MANUAL OF OPHTHALMIC PRACTICE.
Illustrations, crown 8vo, 6s. [LEWIS'S PRACTICAL SERIES.]

BERKELEY HILL, M.B. LOND., F.R.C.S.

Professor of Clinical Surgery in University College; Surgeon to University College Hospital and to the Lock Hospital;

AND

ARTHUR COOPER, L.R.C.P., M.R.C.S.

Surgeon to the Westminster General Dispensary.

SYPHILIS AND LOCAL CONTAGIOUS DISORDERS.
Second Edition, entirely re-written, royal 8vo, 18s.

FROM HOSPITAL WARD TO CONSULTING ROOM,
with Notes by the Way; a Medical Autobiography. By a Graduate of the London University. Post 8vo, 3s. 6d.

SIR W. JENNER, Bart., M.D.

Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales.

THE PRACTICAL MEDICINE OF TO-DAY: Two
Addresses delivered before the British Medical Association, and the Epidemiological Society, (1869). Small 8vo, 1s. 6d.

GEORGE LINDSAY JOHNSON, M.A., M.B., B.C. CANTAB.
Clinical Assistant, late House Surgeon and Chloroformist, Royal Westminster Ophthalmic Hospital, &c.

A NEW METHOD OF TREATING CHRONIC GLAUCOMA, based on Recent Researches into its Pathology. With Illustrations and coloured frontispiece, demy 8vo, 3s. 6d.

H. LEWIS JONES, M.A., M.D., F.R.C.P.

Medical Officer in charge of the Electrical Department in St. Bartholomew's Hospital.

MEDICAL ELECTRICITY. A Practical Handbook for
Students and Practitioners. Second Edition, with Illustrations, crown 8vo, 10s. 6d. [Now ready]
[LEWIS'S PRACTICAL SERIES.]

T. N. KELYNACK, M.D.

Pathologist to the Manchester Royal Infirmary; Demonstrator and Lecturer on Pathology in the Owen's College.

A CONTRIBUTION TO THE PATHOLOGY OF THE VERMIFORM APPENDIX. With Illustrations, large 8vo, 10s. 6d.

HENRY R. KENWOOD, M.B., D.P.H., F.C.S.

Instructor in the Hygienic Laboratory, University College, and Assistant to Professor Corfield in the Public Health Department, University College, &c.

PUBLIC HEALTH LABORATORY WORK.

Second Edition, with Illustrations, cr. 8vo, 10s. 6d. [Now ready.]

[LEWIS'S PRACTICAL SERIES.]

NORMAN KERR, M.D., F.L.S.

President of the Society for the Study of Inebriety; Consulting Physician, Dalrymple Home for Inebriates, &c.

INEBRIETY OR NARCOMANIA: its Etiology, Pathology, Treatment, and Jurisprudence. Third Edition, 8vo, 21s.

NORMAN W. KINGSLEY, M.D.S., D.D.S.

President of the Board of Censors of the State of New York; Member of the American Academy of Dental Science, &c.

A TREATISE ON ORAL DEFORMITIES AS A BRANCH OF MECHANICAL SURGERY. With over 350 Illustrations, 8vo, 16s.

F. CHARLES LARKIN, F.R.C.S. ENG.

Surgeon to the Stanley Hospital; late Assistant Lecturer in Physiology in University College, Liverpool,

AND

RANDLE LEIGH, M.B., B.SC. LOND.

Senior Demonstrator of Physiology in University College, Liverpool.

OUTLINES OF PRACTICAL PHYSIOLOGICAL CHEMISTRY. Second Edition, with Illustrations, crown 8vo, paper 2s. 6d. nett, or cloth 3s. nett.

J. WICKHAM LEGG, F.R.C.P.

Formerly Assistant Physician to Saint Bartholomew's Hospital, and Lecturer on Pathological Anatomy in the Medical School.

ON THE BILE, JAUNDICE, AND BILIOUS DISEASES.
With Illustrations in chromo-lithography, 719 pages, roy. 8vo, 25s.

II.

A GUIDE TO THE EXAMINATION OF THE URINE.
Seventh Edition, edited and revised, by H. LEWIS JONES, M.D., M.A., F.R.C.P., Medical Officer in charge of the Electrical Department in St. Bartholomew's Hospital. With Illustrations, fcap. 8vo, 3s. 6d.

ARTHUR H. N. LEWERS, M.D. LOND., M.R.C.P. LOND.

Obstetric Physician to the London Hospital; Examiner in Midwifery at the Conjoint Board of the Royal College of Physicians, London, and the Royal College of Surgeons, England.

A PRACTICAL TEXTBOOK OF THE DISEASES OF WOMEN. Fourth Edition, Illustrations, crown 8vo, 10s. 6d.

[LEWIS'S PRACTICAL SERIES.]

LEWIS'S POCKET MEDICAL VOCABULARY.

Second Edition, thoroughly revised, 32mo, roan, 3s. 6d.

T. R. LEWIS, M.B., F.R.S. ELECT, ETC.

Late Fellow of the Calcutta University, Surgeon-Major Army Medical Staff, &c.

PHYSIOLOGICAL & PATHOLOGICAL RESEARCHES.

Arranged and edited by SIR WM. AITKEN, M.D., F.R.S., G. E. DOBSON, M.B., F.R.S., and A. E. BROWN, B.Sc. Crown 4to, portrait, 5 maps, 43 plates and 67 wood engravings, 30s. nett.

*. * A few copies only of this work remain for sale.

ROBERT LINDSAY, A.M., M.B., F.R.C.S.E.

Retired Surgeon, Army Medical Department.

AN ESSAY ON MALARIA AND ITS CONSEQUENCES.

Crown 8vo, 4s.

C. B. LOCKWOOD, F.R.C.S.

Hunterian Professor, Royal College of Surgeons of England; Surgeon to the Great Northern Hospital; Senior Demonstrator of Anatomy and Operative Surgery in St. Bartholomew's Hospital.

HUNTERIAN LECTURES ON THE MORBID ANATOMY, PATHOLOGY AND TREATMENT OF HERNIA.

36 Illustrations, demy 8vo, 5s.

WILLIAM THOMPSON LUSK, A.M., M.D.

Professor of Obstetrics and Diseases of Women in the Bellevue Hospital Medical College, &c.

THE SCIENCE AND ART OF MIDWIFERY.

Fourth Edition, with numerous Illustrations, 8vo, 18s.

A. W. MACFARLANE, M.D., F.R.C.P. EDIN.

Examiner in Medical Jurisprudence in the University of Glasgow; Honorary Consulting Physician (late Physician) Kilmarnock Infirmary.

INSOMNIA AND ITS THERAPEUTICS.

Medium 8vo, 12s. 6d.

RAWDON MACNAMARA.

Professor of Materia Medica, Royal College of Surgeons, Ireland; Senior Surgeon to the Westmoreland (Lock) Government Hospital; Surgeon to the Meath Hospital, &c.

AN INTRODUCTION TO THE STUDY OF THE BRITISH PHARMACOPŒIA. Demy 32mo, 1s. 6d.

JOHN MACPHERSON, M.D.

Inspector-General of Hospitals H.M. Bengal Army (Retired).

ANNALS OF CHOLERA FROM THE EARLIEST PERIODS TO THE YEAR 1817. With a map. Demy 8vo, 7s. 6d.

A. COWLEY MALLEY, B.A., M.B., E.CH., T.C.D.

PHOTO-MICROGRAPHY; including a description of the Wet Collodion and Gelatino-Bromide Processes, together with the best methods of Mounting and Preparing Microscopic Objects for Photo-Micrography. Second Edition, with Photographs and Illustrations, crown 8vo, 7s. 6d.

PATRICK MANSON, M.D., C.M.

THE FILARIA SANGUINIS HOMINIS; AND CERTAIN NEW FORMS OF PARASITIC DISEASE IN INDIA, CHINA, AND WARM COUNTRIES. Illustrated with Plates and Charts. 8vo, 10s. 6d.

JEFFERY A. MARSTON, M.D., C.B., F.R.C.S., M.R.C.P. LOND.
Surgeon General Medical Staff (Retired).

NOTES ON TYPHOID FEVER: Tropical Life and its Sequelæ. Crown 8vo, 3s. 6d.

EDWARD MARTIN, A.M., M.D.

MINOR SURGERY AND BANDAGING WITH AN APPENDIX ON VENEREAL DISEASES. 82 Illustrations, crown 8vo, 4s.

WILLIAM MARTINDALE, F.C.S.

Late Examiner of the Pharmaceutical Society, and late Teacher of Pharmacy and Demonstrator of Materia Medica at University College,

AND

W. WYNN WESTCOTT, M.B. LOND.

Coroner for North-East London.

THE EXTRA PHARMACOPŒIA, with Medical References, and a Therapeutic Index of Diseases and Symptoms. Eighth Edition, limp roan, med. 24mo, 9s. [Just published.]

WILLIAM MARTINDALE, F.C.S.

Late Examiner of the Pharmaceutical Society, &c.

I.
COCA, AND COCAINE: their History, Medical and Economic Uses, and Medicinal Preparations. Fourth Edition, fcap. 8vo, 2s. [Now ready.]

II.
ANALYSES OF TWELVE THOUSAND PRESCRIPTIONS: being Statistics of the Frequency of Use therein of all Official and Unofficial Preparations. Fcap. 4to, 2s. 6d. *nett*.

MATERIA MEDICA LABELS.

Adapted for Public and Private Collections. Compiled from the British Pharmacopœia of 1885, with the additions of 1890. The Labels are arranged in Two Divisions:—

Division I.—Comprises, with few exceptions, Substances of Organized Structure, obtained from the Vegetable and Animal Kingdoms.

Division II.—Comprises Chemical Materia Medica, including Alcohols, Alkaloids, Sugars, and Neutral Bodies.

On plain paper, 10s. 6d. *nett*. On gummed paper, 12s. 6d. *nett*.

The 24 additional Labels of 1890 only, 1s. *nett*.

* Specimens of the Labels, of which there are over 470, will be sent on application.

S. E. MAUNSELL, L.R.C.S.I.

Surgeon-Major, Medical Staff.

NOTES OF MEDICAL EXPERIENCES IN INDIA
PRINCIPALLY WITH REFERENCE TO DISEASES OF THE
EYE. With Map, post 8vo, 3s. 6d.

ANGEL MONEY, M.D. LOND., F.R.C.P.

Late Assistant Physician to University College Hospital, and to the Hospital for Sick Children. Great Ormond Street.

THE STUDENT'S TEXTBOOK OF THE PRACTICE
OF MEDICINE. Fcap. 8vo, 6s. 6d.

A. STANFORD MORTON, M.B., F.R.C.S. ENG.

Assistant Surgeon to the Moorfields Ophthalmic Hospital; Ophthalmic Surgeon to the Great Northern Central Hospital.

REFRACTION OF THE EYE: Its Diagnosis, and the
Correction of its Errors. Sixth Edition, with Illustrations, small 8vo,
3s. 6d. [Just ready.]

C. W. MANSELL MOULLIN, M.A., M.D. OXON., F.R.C.S.

Surgeon to and Lecturer on Physiology at the London Hospital; late Radcliffe's Travelling Fellow and Fellow of Pembroke College, Oxford, &c.

ENLARGEMENT OF THE PROSTATE: its Treatment
and Radical Cure. With plates, 8vo, 6s. [Now ready.]

SPRAINS; THEIR CONSEQUENCES AND TREAT-
MENT. Second Edition, crown 8vo, 4s. 6d.

PAUL F. MUNDE, M.D.

Professor of Gynecology at the New York Polyclinic; President of the New York Obstetrical Society and Vice-President of the British Gynecological Society, &c.

THE MANAGEMENT OF PREGNANCY, PARTURI-
TION, AND THE PUERPERAL STATE. Second Edition, square
8vo, 3s. 6d.

WILLIAM MURRAY, M.D., F.R.C.P. LOND.

ROUGH NOTES ON REMEDIES. Second Edition, crown 8vo,
3s. 6d. [Now ready.]

ILLUSTRATIONS OF THE INDUCTIVE METHOD IN
MEDICINE. Crown 8vo, 3s. 6d.

WILLIAM MURRELL, M.D., F.R.C.P.

Physician to, and Lecturer on Pharmacology and Therapeutics at Westminster Hospital; late Examiner in Materia Medica to the Royal College of Physicians of London, &c.

WHAT TO DO IN CASES OF POISONING.
Eighth Edition, royal 32mo, 3s. 6d. [Now ready.]

MASSOTHERAPEUTICS, OR MASSAGE AS A MODE
OF TREATMENT. Fifth Edition, with Illustrations, crown 8vo, 4s. 6d.

HENRY D. NOYES, A.M., M.D.

Professor of Ophthalmology and Otolaryngology in Bellevue Hospital Medical College; Executive Surgeon to the New York Eye and Ear Infirmary, &c.

A TEXTBOOK ON DISEASES OF THE EYE.

Second and revised Edition, Illustrated by 5 chromo-lithographic plates, 10 plates in black and colours and 269 wood engravings, 28s. *nett*.

GEORGE OLIVER, M.D., F.R.C.P.

I.

PULSE-GAUGING: A Clinical Study of Radial Measurement and Pulse Pressure. With Illustrations, fcap. 8vo, 3s. 6d.

[*Now ready.*]

II.

THE HARROGATE WATERS: Data Chemical and Therapeutical, with notes on the Climate of Harrogate. Addressed to the Medical Profession. With Map of the Wells, crown 8vo, 3s. 6d.

III.

ON BEDSIDE URINE TESTING: a Clinical Guide to the Observation of Urine in the course of Work. Fourth Edition, fcap. 8vo, 3s. 6d.

DR. A. ONODI.

Lecturer on Rhino-Laryngology in the University of Budapest.

THE ANATOMY OF THE NASAL CAVITY, AND ITS ACCESSORY SINUSES. An Atlas for Practitioners and Students, translated by ST CLAIR THOMSON, M.D. LOND., F.R.C.S. ENG., M.R.C.P. LOND. Plates, small 4to, 6s. *nett*. [*Now ready.*]

SAM. OSBORN, F.R.C.S.

Chief Surgeon to the Metropolitan Corps of the St. John Ambulance Brigade; Surgeon to the Hospital for Women, Soho Square, &c.

I.

AMBULANCE LECTURES: FIRST AID TO THE INJURED. Third Edition, with Illustrations, fcap. 8vo, 2s.

II.

AMBULANCE LECTURES: HOME NURSING AND HYGIENE. Third Edition, with Illustrations, fcap. 8vo, 2s.

[*Just ready.*]

WILLIAM OSLER, M.D., F.R.C.P. LOND.

President of the Association of American Physicians; Professor of Medicine, Johns Hopkins University, and Physician-in-Chief Johns Hopkins Hospital, Baltimore.

I.

ON CHOREA AND CHOREIFORM AFFECTIONS. Large 8vo, 5s.

II.

THE CEREBRAL PALSIES OF CHILDREN. A Clinical Study from the Infirmary for Nervous Diseases, Philadelphia. Demy 8vo, 5s.

KURRE W. OSTROM.

Instructor in Massage and Swedish Movements in the Philadelphia Polyclinic and College for Graduates in Medicine.

MASSAGE AND THE ORIGINAL SWEDISH MOVEMENTS; their application to various diseases of the body. Third Edition, with Illustrations, 12mo, 3s. 6d. *nett*.

CHARLES A. PARKER. F.R.C.S. EDIN.

Assistant Surgeon to the Hospital for Diseases of the Throat, Golden Square, London.

POST-NASAL GROWTHS. Demy 8vo, 4s. 6d. [*Just published*]

ROBERT W. PARKER.

Senior Surgeon to the East London Hospital for Children; Surgeon to the German Hospital.

DIPHTHERIA: ITS NATURE AND TREATMENT,
WITH SPECIAL REFERENCE TO THE OPERATION, AFTER-
TREATMENT AND COMPLICATIONS OF TRACHEOTOMY.
Third Edition, with Illustrations, 8vo, 6s.

CONGENITAL CLUB-FOOT; ITS NATURE AND TREATMENT. With special reference to the subcutaneous division of Tarsal Ligaments. 8vo, 7s. 6d.

LOUIS C. PARKES, M.D., D.P.H. LOND. UNIV.

Lecturer on Public Health at St. George's Hospital Medical School; Medical Officer of Health for Chelsea.

HYGIENE AND PUBLIC HEALTH. Fourth Edition, with numerous Illustrations, crown 8vo, 10s. 6d.

[LEWIS'S PRACTICAL SERIES.]

INFECTIOUS DISEASES, NOTIFICATION AND PREVENTION. Fcap. 8vo, cloth, 2s. 6d; roan, 4s. 6d.

JOHN S. PARRY, M.D.

Obstetrician to the Philadelphia Hospital, Vice-President of the Obstetrical and Pathological Societies of Philadelphia, &c.

EXTRA-UTERINE PREGNANCY; Its Causes, Species,
Pathological Anatomy, Clinical History, Diagnosis, Prognosis and Treatment. 8vo, 8s.

E. RANDOLPH PEASLEE, M.D., LL.D.

Late Professor of Gynæcology in the Medical Department of Dartmouth College; President of New York Academy of Medicine, &c., &c.

OVARIAN TUMOURS: Their Pathology, Diagnosis, and Treatment, especially by Ovariectomy. Illustrations, roy. 8vo, 16s.

LESLIE PHILLIPS, M.D.

Surgeon to the Birmingham and Midland Skin and Lock Hospital.

MEDICATED BATHS IN THE TREATMENT OF SKIN DISEASES. Crown 8vo, 4s. 6d.

HENRY G. PIFFARD, A.M., M.D.

Clinical Professor of Dermatology, University of the City of New York; Surgeon in Charge of the New York Dispensary for Diseases of the Skin, &c.

A PRACTICAL TREATISE ON DISEASES OF THE SKIN. With 50 full page Original Plates and 33 Illustrations in the Text, 4to, £2 12s. 6d. nett.

G. V. POORE, M.D., F.R.C.P.

Professor of Medical Jurisprudence, University College; Assistant Physician to, and Physician in charge of the Throat Department of, University College Hospital.

LECTURES ON THE PHYSICAL EXAMINATION OF THE MOUTH AND THROAT. With an Appendix of Cases. 8vo, 3s. 6d.

R. DOUGLAS POWELL, M.D., F.R.C.P.

Physician Extra-ordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption and Diseases of the Chest at Brompton.

DISEASES OF THE LUNGS AND PLEURÆ, INCLUDING CONSUMPTION. Fourth Edition, with coloured plates and wood engravings, 8vo, 18s.

TABLE OF PHYSICAL EXAMINATION OF THE LUNGS: with Note on International Nomenclature of Physical Signs (reprinted from DR. POWELL's "Diseases of the Lungs,"). On one sheet, 6d.

HERBERT A. POWELL, M.A., M.D., M.CH. OXON., F.R.C.S.E.

THE SURGICAL ASPECT OF TRAUMATIC INSANITY. Medium 8vo, 2s. 6d.

D'ARCY POWER, M.A., M.B. OXON., F.R.C.S. ENG.

Demonstrator of Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; Examiner in the University of Durham; Member of the Conjoint Examining Board of the Royal College of Physicians (Lond.) and of Surgeons (Eng.)

THE SURGICAL DISEASES OF CHILDREN AND THEIR TREATMENT BY MODERN METHODS. With Illustrations, crown 8vo, 10s. 6d. [Now ready.]

[LEWIS'S PRACTICAL SERIES.]

URBAN PRITCHARD, M.D. EDIN., F.R.C.S. ENG.

Professor of Aural Surgery at King's College, London; Aural Surgeon to King's College Hospital; Senior Surgeon to the Royal Ear Hospital.

HANDBOOK OF DISEASES OF THE EAR FOR THE USE OF STUDENTS AND PRACTITIONERS. Third Edition, with Illustrations, crown 8vo, 6s. [Now ready.]

[LEWIS'S PRACTICAL SERIES.]

CHARLES W. PURDY, M.D. (QUEEN'S UNIV.)

Professor of Genito-Urinary and Renal Diseases in the Chicago Polyclinic, &c., &c.

BRIGHT'S DISEASE AND THE ALLIED AFFECTIONS OF THE KIDNEYS. With Illustrations, large 8vo, 8s. 6d.

DR. THEODOR PUSCHMANN.

Public Professor in Ordinary at the University of Vienna.

A HISTORY OF MEDICAL EDUCATION FROM THE MOST REMOTE TO THE MOST RECENT TIMES. Translated and edited by EVAN H. HARE, M.A. OXON., F.R.C.S. ENG., L.S.A. Demy 8vo, 21s.

C. H. RALFE, M.A., M.D. CANTAB., F.R.C.P. LOND.

Assistant Physician to the London Hospital; Examiner in Medicine to the University of Durham, &c., &c.

A PRACTICAL TREATISE ON DISEASES OF THE KIDNEYS AND URINARY DERANGEMENTS. With Illustrations, crown 8vo, 10s. 6d. [LEWIS'S PRACTICAL SERIES.]

FRANCIS H. RANKIN, M.D.

President of the New York Medical Society.

HYGIENE OF CHILDHOOD. Suggestions for the care of Children after the Period of Infancy to the completion of Puberty. Crown 8vo, 3s.

AMBROSE L. RANNEY, A.M., M.D.

Professor of the Anatomy and Physiology of the Nervous System in the New York Post-Graduate Medical School and Hospital, &c.

THE APPLIED ANATOMY OF THE NERVOUS SYSTEM. Second Edition, 238 Illustrations, large 8vo, 21s.

H. A. REEVES, F.R.C.S. EDIN.

Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital; Surgeon to the Royal Orthopædic Hospital.

BODILY DEFORMITIES AND THEIR TREATMENT:
A HANDBOOK OF PRACTICAL ORTHOPÆDICS. Illustrations,
crown 8vo, 8s. 6d. [LEWIS'S PRACTICAL SERIES.]

RALPH RICHARDSON, M.A., M.D.

Fellow of the College of Physicians, Edinburgh.

ON THE NATURE OF LIFE: An Introductory Chapter to Pathology. Second edition, revised and enlarged. Fcap. 4to, 10s. 6d.

SAMUEL RIDEAL, D.SC. (LOND.), F.I.C., F.C.S., F.G.S.

Fellow of University College, London.

I.

PRACTICAL ORGANIC CHEMISTRY; The Detection and Properties of some of the more important Organic Compounds. 12mo, 2s. 6d.

II.

PRACTICAL CHEMISTRY FOR MEDICAL STUDENTS, required at the First Examination of the Conjoint Examining Board in England. Fcap 8vo, 2s.

J. JAMES RIDGE, M.D.

Medical Officer of Health, Enfield.

ALCOHOL AND PUBLIC HEALTH. Second Edition, crown 8vo, 2s.

E. A. RIDSDALE.

Associate of the Royal School of Mines.

COSMIC EVOLUTION; being Speculations on the Origin of our Environment. Fcap. 8vo, 3s.

SYDNEY RINGER, M.D., F.R.S.

Holme Professor of Clinical Medicine in University College; Physician to University College Hospital.

I.

A HANDBOOK OF THERAPEUTICS.

Thirteenth Edition, thoroughly revised, 8vo.

[In preparation.

II.

**ON THE TEMPERATURE OF THE BODY AS
A MEANS OF DIAGNOSIS AND PROGNOSIS IN PHTHISIS.**

Second Edition, small 8vo, 2s. 6d.

R. LAWTON ROBERTS, M.D. LOND., D.P.H. CAMB., M.R.C.S. ENG.
Honorary Life Member of, and Lecturer and Examiner to, the St. John Ambulance Association.

I.

ILLUSTRATED LECTURES ON AMBULANCE WORK.

Fifth Edition, copiously Illustrated, crown 8vo, 2s. 6d.

[Just published.

II.

**ILLUSTRATED LECTURES ON NURSING AND HY-
GIENE.** Second Edition, with Illustrations, crown 8vo, 2s. 6d.

FREDERICK T. ROBERTS, M.D., B.SC., F.R.C.P.

Professor of the Principles and Practice of Medicine in University College; Physician to University College Hospital; Consulting Physician to Brompton Consumption Hospital, &c.

I.

THE THEORY AND PRACTICE OF MEDICINE.

Ninth Edition, with Illustrations, large 8vo, 21s.

II.

THE OFFICINAL MATERIA MEDICA.

Second Edition, entirely rewritten in accordance with the latest British Pharmacopœia, fcap. 8vo, 7s. 6d.

III.

**NOTES ON THE ADDITIONS MADE TO THE BRITISH
PHARMACOPŒIA, 1890.** Fcap. 8vo, 1s.

A. D. ROCKWELL, A.M., M.D.

Formerly Professor of Electro-Therapeutics in the New York Post Graduate Medical School and Hospital; Fellow of the New York Academy of Medicine, &c.

**THE MEDICAL AND SURGICAL USES OF ELEC-
TRICITY.** New Edition, illustrated with 200 Engravings, large 8vo,
18s. nett.

[Just Published.

H. D. ROLLESTON, M.D., F.R.C.P.

[See Cambridge Natural Science Manuals, page 5.

D. B. ST. JOHN ROOSA, M.D.

Consulting Surgeon to the Brooklyn Eye and Ear Hospital, &c.

**A PRACTICAL TREATISE ON THE DISEASES OF
THE EAR:** Including a Sketch of Aural Anatomy and Physiology.
Seventh Edition, Illustrated, large 8vo, 25s.

ROBSON ROOSE, M.D., LL.D., F.C.S.
Fellow of the Royal College of Physicians in Edinburgh, &c.

**GOUT, AND ITS RELATIONS TO DISEASES OF
 THE LIVER AND KIDNEYS.** Seventh Edition, crown 8vo, 4s. 6d.
 [Now ready.]

**NERVE PROSTRATION AND OTHER FUNCTIONAL
 DISORDERS OF DAILY LIFE.** Second Edition, demy 8vo, 18s.

**LEPROSY AND ITS PREVENTION: as Illustrated by
 Norwegian Experience.** Crown 8vo, 3s. 6d.

WILLIAM ROSE, M.B., B.S. LOND., F.R.C.S.
Professor of Surgery in King's College, London, and Surgeon to King's College Hospital.
HARELIP AND CLEFT PALATE. With Illustrations, demy
 8vo, 6s.

BERNARD ROTH, F.R.C.S.
*Fellow of the Medical Society of London; Member of the Clinical and Pathological Societies
 and of the Medical Officers of Schools' Association.*
**THE TREATMENT OF LATERAL CURVATURE OF
 THE SPINE.** With Photographic and other Illustrations, demy 8vo,
 5s.

W. H. O. SANKEY, M.D. LOND., F.R.C.P.
Late Lecturer on Mental Diseases, University College, London, etc.
LECTURES ON MENTAL DISEASE. Second Edition, with
 coloured Plates, 8vo, 12s. 6d.

JOHN SAVORY.
Member of the Society of Apothecaries, London.
**A COMPENDIUM OF DOMESTIC MEDICINE AND
 COMPANION TO THE MEDICINE CHEST:** Intended as a
 source of easy reference for Clergymen, Master Mariners, and Tra-
 vellers; and for Families resident at a distance from professional assist-
 ance. Tenth Edition, sm. 8vo, 5s.

DR. C. SCHIMMELBUSCH.
Privat-docent and Assistant Surgeon in Prof. v. Bergmann's University Clinic at Berlin.
THE ASEPTIC TREATMENT OF WOUNDS.
 With a Preface by Professor BERGMANN. Translated from the Second
 German Edition by A. T. RAKE, M.B., B.S. LOND., F.R.C.S. ENG.,
 Registrar and Pathologist to the East London Hospital for Children.
 With Illustrations, crown 8vo, 5s. [Now ready.]

E. SCHMIEGELOW, M.D.
*Consulting Physician in Laryngology to the Municipal Hospital and Director of the Oto-
 Laryngological Department in the Polyclinic at Copenhagen.*
ASTHMA: Especially in its Relation to Nasal Disease.
 Demy 8vo, 4s. 6d.

DR. B. S. SCHULTZE.

Professor of Gynecology ; Director of the Lying-in Hospital, and of the Gynecological Clinic at Jena.

THE PATHOLOGY AND TREATMENT OF DISPLACEMENTS OF THE UTERUS. Translated by J. J. MACAN, M.A., M.R.C.S. and edited by A. V. MACAN, M.B., M.Ch., Master of the Rotunda Lying-in Hospital, Dublin. With 120 Illustrations, medium 8vo, 12s. 6d.

A. C. SEWARD, M.A., F.G.S.

[See Cambridge Natural Science Manuals, page 5.]

JOHN SHAW, M.D. LOND., M.R.C.P.

Obstetric Physician to the North-West London Hospital.

ANTISEPTICS IN OBSTETRIC NURSING. A Text-book for Nurses on the Application of Antiseptics to Gynæcology and Midwifery. Coloured plate and woodcuts, 8vo, 3s. 6d.

G. E. SHUTTLEWORTH, B.A., M.D.

Late Medical Superintendent, Royal Albert Asylum for Idiots and Imbeciles of the Northern Counties, Lancaster ; formerly Assistant Medical Officer, Earlswood Asylum.

MENTALLY-DEFICIENT CHILDREN THEIR TREATMENT AND TRAINING. With Illustrations, crown 8vo, 4s.

A. J. C. SKENE, M.D.

Professor of Gynecology in the Long Island College Hospital, Brooklyn, New York.

TREATISE ON THE DISEASES OF WOMEN, FOR THE USE OF STUDENTS AND PRACTITIONERS. Second Edition, with coloured plates and 251 engravings, large 8vo, 28s.

J. LEWIS SMITH, M.D.

Physician to the New York Foundling Asylum ; Clinical Professor of Diseases of Children in Bellevue Hospital Medical College.

A TREATISE ON THE DISEASES OF INFANCY AND CHILDHOOD. Seventh Edition, with Illustrations, large 8vo, 21s.

FRANCIS W. SMITH, M.B., B.S.

THE SALINE WATERS OF LEAMINGTON. Second Edit., with Illustrations, crown 8vo, 1s. *nett.*

E. HUGH SNELL, M.D., B.SC. LOND.

Diplomate in Public Health of the University of Cambridge ; London County Council Medical Officer to the Blackwall Tunnel.

COMPRESSED AIR ILLNESS, OR SO-CALLED CAISON DISEASE. With Illustrations, demy 8vo, 10s. 6d. [Now ready.]

JOHN KENT SPENDER, M.D. LOND.

Physician to the Royal Mineral Water Hospital, Bath.

THE EARLY SYMPTOMS AND THE EARLY TREATMENT OF OSTEO-ARTHRITIS, commonly called Rheumatoid Arthritis, with special reference to the Bath Thermal Waters. Sm. 8vo, 2s. 6d.

LOUIS STARR, M.D.

Physician to the Children's Hospital, Philadelphia; late Clinical Professor of Diseases of Children in the Hospital of the University of Pennsylvania.

HYGIENE OF THE NURSERY. Including the General Regimen and Feeding of Infants and Children; Massage, and the Domestic Management of the Ordinary Emergencies of Early Life. Fifth Edition, with Illustrations, crown 8vo, 3s. 6d.

JAMES STARTIN, M.B., M.R.C.S.

Senior Surgeon to the London Skin Hospital.

LECTURES ON THE PARASITIC DISEASES OF THE SKIN. VEGETOID AND ANIMAL. With Illustrations, crown 8vo, 2s. 6d.

JOHN LINDSAY STEVEN, M.D.

Assistant Physician and Pathologist, Glasgow Royal Infirmary; Physician for Out-patients, Royal Hospital for Sick Children, Glasgow; Lecturer on Pathology, St. Mungo's and Queen Margaret Colleges, Glasgow, &c.

THE PATHOLOGY OF MEDIASTINAL TUMOURS. With special reference to Diagnosis. With Plates, 8vo, 4s. 6d.

LEWIS A. STIMSON, B.A., M.D.

Surgeon to the Presbyterian and Bellevue Hospitals; Professor of Clinical Surgery in the Medical Faculty of the University of the City of New York, &c.

A MANUAL OF OPERATIVE SURGERY.

Second Edition, with three hundred and forty-two Illustrations, post 8vo, 10s. 6d.

JAMES STOCKEN, L.D.S. ENG.

Pereira Prizeman for Materia Medica; Late Dental Surgeon to the National Dental Hospital.

DENTAL MATERIA MEDICA AND THERAPEUTICS.

Fourth edition, revised by LESLIE M. STOCKEN, L.R.C.P., M.R.C.S., L.D.S., and J. O. BUTCHER, L.D.S. ENG., Assistant Dental Surgeon to Guy's Hospital. Fcap. 8vo, 4s. [Now ready.]

ADOLF STRÜMPPELL.

Professor and Director of the Medical Clinique at Erlangen.

A TEXT-BOOK OF MEDICINE FOR STUDENTS AND PRACTITIONERS. Second Edition translated from the German

by Dr. H. F. VICKERY and Dr. P. C. KNAPP, with Editorial Notes by Dr. F. C. SHATTUCK, Visiting Physician to the Massachusetts General Hospital, etc. Complete in one large vol., with 119 Illustrations, imp. 8vo, 28s.

JUKES DE STYRAP, M.R.C.P.I., ETC.

Physician-Extraordinary, late Physician in Ordinary, to the Salop Infirmary; Consulting Physician to the South Salop and Montgomeryshire Infirmaries, etc.

I.

THE YOUNG PRACTITIONER: WITH PRACTICAL HINTS AND INSTRUCTIVE SUGGESTIONS, AS SUBSIDIARY AIDS, FOR HIS GUIDANCE ON ENTERING INTO PRIVATE PRACTICE. Demy 8vo, 7s. 6d. *nett.*

II.

A CODE OF MEDICAL ETHICS: WITH GENERAL AND SPECIAL RULES FOR THE GUIDANCE OF THE FACULTY AND THE PUBLIC IN THE COMPLEX RELATIONS OF PROFESSIONAL LIFE. Fourth Edition, demy 8vo, 3s. 6d. *nett.*

III.

MEDICO-CHIRURGICAL TARIFFS.

Fifth Edition, revised and enlarged, fcap. 4to, 2s. *nett.*

IV.

THE YOUNG PRACTITIONER: HIS CODE AND TARIFF. Being the above three works in one volume. Demy 8vo, 10s. 6d. *nett.*

C. W. SUCKLING, M.D. LOND., M.R.C.P.

Professor of Materia Medica and Therapeutics at the Queen's College, Physician to the Queen's Hospital, Birmingham, etc.

I.

ON THE DIAGNOSIS OF DISEASES OF THE BRAIN, SPINAL CORD, AND NERVES. With Illustrations, crown 8vo, 8s. 6d.

II.

ON THE TREATMENT OF DISEASES OF THE NERVOUS SYSTEM. Crown 8vo, 7s. 6d.

JOHN BLAND SUTTON, F.R.C.S.

Lecturer on Comparative Anatomy, Senior Demonstrator of Anatomy, and Assistant Surgeon to the Middlesex Hospital; Erasmus Wilson Lecturer, Royal College of Surgeons, England.

LIGAMENTS: THEIR NATURE AND MORPHOLOGY.

Second Edition, with numerous Illustrations, post 8vo. [*Just ready.*]

HENRY R. SWANZY, A.M., M.B., F.R.C.S.I.

Surgeon to the National Eye and Ear Infirmary, and Ophthalmic Surgeon to the Adelaide Hospital, Dublin.

A HANDBOOK OF THE DISEASES OF THE EYE AND THEIR TREATMENT. Fifth Edition, edited under supervision of the Author by LOUIS WERNER, M.B., B.Ch., Examiner in Ophthalmic Surgery in the University of Dublin, and in the Royal University of Ireland. Illustrated with wood-engravings, colour tests, etc., small 8vo, 10s. 6d.

EUGENE S. TALBOT, M.D., D.D.S.

Professor of Dental Surgery in the Woman's Medical College; Lecturer on Dental Pathology and Surgery in Rush Medical College, Chicago.

IRREGULARITIES OF THE TEETH AND THEIR TREATMENT. With 152 Illustrations, royal 8vo, 10s. 6d.

ALBERT TAYLOR.

Associate Sanitary Institute; Chief Sanitary Inspector to the Vestry of St. George, Hanover Square, etc.

THE SANITARY INSPECTOR'S HANDBOOK.

With Illustrations, cr. 8vo., 5s.

H. COUPLAND TAYLOR, M.D.

Fellow of the Royal Meteorological Society.

**WANDERINGS IN SEARCH OF HEALTH, OR
MEDICAL AND METEOROLOGICAL NOTES ON VARIOUS
FOREIGN HEALTH RESORTS.** With Illustrations, crown 8vo, 6s.

J. C. THOROWGOOD, M.D.

Assistant Physician to the City of London Hospital for Diseases of the Chest.

**THE CLIMATIC TREATMENT OF CONSUMPTION
AND CHRONIC LUNG DISEASES.** Third Edition, post 8vo, 3s. 6d.

D. HACK TUKE, M.D., LL.D.

Fellow of the Royal College of Physicians, London.

**THE INSANE IN THE UNITED STATES AND
CANADA.** Demy 8vo, 7s. 6d.

DR. R. ULTZMANN.

ON STERILITY AND IMPOTENCE IN MAN. Translated
from the German with notes and additions by ARTHUR COOPER, L.R.C.P.,
M.R.C.S., Surgeon to the Westminster General Dispensary. With
Illustrations, fcap. 8vo, 2s. 6d.

W. H. VAN BUREN, M.D., LL.D.

Professor of Surgery in the Bellevue Hospital Medical College.

**DISEASES OF THE RECTUM: And the Surgery of the
Lower Bowel.** Second Edition, with Illustrations, 8vo, 14s.

ALFRED VOGEL, M.D.

Professor of Clinical Medicine in the University of Dorpat, Russia.

**A PRACTICAL TREATISE ON THE DISEASES OF
CHILDREN.** Third Edition, translated and edited by H. RAPHAEL,
M.D., from the Eighth German Edition, illustrated by six lithographic
plates, part coloured, royal 8vo, 18s.

A. DUNBAR WALKER, M.D., C.M.

THE PARENT'S MEDICAL NOTE BOOK.

Oblong post 8vo, cloth, 1s. 6d.

A. J. WALL, M.D. LOND.

Fellow of the Royal College of Surgeons of England; of the Medical Staff of H. M. Indian Army (Retired List).

ASIATIC CHOLERA: its History, Pathology, and Modern Treatment. Demy 8vo, 6s.

JOHN RICHARD WARDELL, M.D. EDIN., F.R.C.P. LOND.

Late Consulting Physician to the General Hospital Tunbridge Wells.

CONTRIBUTIONS TO PATHOLOGY AND THE PRACTICE OF MEDICINE. Medium 8vo, 21s.

W. SPENCER WATSON, B.M. LOND., F.R.C.S. ENG.

Surgeon to the Throat Department of the Great Northern Hospital; Senior Surgeon to the Royal South London Ophthalmic Hospital.

I.
DISEASES OF THE NOSE AND ITS ACCESSORY CAVITIES. Second Edition, with Illustrations, demy 8vo, 12s. 6d.

II.
THE ANATOMY AND DISEASES OF THE LACHRYMAL PASSAGES. With Illustrations, demy 8vo, 2s. 6d.

III.
EYEBALL-TENSION: Its Effects on the Sight and its Treatment. With woodcuts, p. 8vo, 2s. 6d.

IV.
ON ABSCESS AND TUMOURS OF THE ORBIT. Post 8vo, 2s. 6d.

FRANCIS H. WELCH, F.R.C.S.

Surgeon Major, A.M.D.

ENTERIC FEVER: as Illustrated by Army Data at Home and Abroad, its Prevalence and Modifications, Ætiology, Pathology and Treatment. 8vo, 5s. 6d.

W. WYNN WESTCOTT, M.B.

Deputy Coroner for Central Middlesex.

SUICIDE; its History, Literature, Jurisprudence, and Prevention. Crown 8vo, 6s.

FRANK J. WETHERED, M.D.

Medical Registrar to the Middlesex Hospital, and Demonstrator of Practical Medicine in the Middlesex Hospital Medical School; late Assistant Physician to the City of London Chest Hospital, Victoria Park.

MEDICAL MICROSCOPY. A Guide to the Use of the Microscope in Medical Practice. With Illustrations, crown 8vo, 9s.
[LEWIS'S PRACTICAL SERIES.]

W. C. D. WHETHAM, M.A.

[See Cambridge Natural Science Manuals, page 5.]

E. G. WHITTLE, M.D. LOND., F.R.C.S. ENG.

Senior Surgeon to the Royal Alexandra Hospital for Sick Children, Brighton.

CONGESTIVE NEURASTHENIA, OR INSOMNIA AND NERVE DEPRESSION. Crown 8vo, 3s. 6d.

L. R. WILBERFORCE, M.A.

AND

T. C. FITZPATRICK, M.A.

Demonstrators at the Cavendish Laboratory, Cambridge.

A LABORATORY NOTE-BOOK OF ELEMENTARY PRACTICAL PHYSICS.

I. MECHANICS AND HYDROSTATICS. Quarto, paper covers, 1s.

SIR JOHN WILLIAMS, BART., M.D., F.R.C.P.

Consulting Physician to University College Hospital; Physician Accoucheur to H.R.H. Princess Beatrice, &c.

CANCER OF THE UTERUS: Being the Harveian Lectures for 1886. Illustrated with Lithographic Plates, royal 8vo, 10s. 6d.

R. T. WILLIAMSON, M.D. LOND., M.R.C.P.

Medical Registrar, Royal Infirmary, and Assistant in Medicine, Owen's College, Manchester

ON THE RELATION OF DISEASES OF THE SPINAL CORD TO THE DISTRIBUTION AND LESIONS OF THE SPINAL BLOOD VESSELS. Royal 8vo, 2s.

E. T. WILSON, M.B. OXON., F.R.C.P. LOND.

Physician to the Cheltenham General Hospital; Associate Metropolitan Association of Medical Officers of Health.

DISINFECTANTS AND ANTISEPTICS: HOW TO USE THEM. In Packets of one doz. price 1s., by post 1s. 1d.

[Just thoroughly revised.]

DR. F. WINCKEL.

Formerly Professor and Director of the Gynæcological Clinic at the University of Rostock.

THE PATHOLOGY AND TREATMENT OF CHILD-BED: A Treatise for Physicians and Students. Translated from the Second German Edition, with many additional notes by the Author, by J. R. CHADWICK, M.D. 8vo, 14s.

BERTRAM C. A. WINDLE, D.SC., M.D., M.A. DUBL.

Professor of Anatomy in Mason College, Birmingham; sometime Examiner in Anatomy in the Universities of Cambridge, Aberdeen, and Durham.

A HANDBOOK OF SURFACE ANATOMY AND LAND-MARKS. Second Edition, revised and enlarged, in collaboration with T. MANNERS-SMITH, M.A. (Cantab.), M.R.C.S., Lecturer on Osteology, Mason College, Birmingham. Illustrated with plain and coloured figures, post 8vo, 3s. 6d.

[Now ready.]

EDWARD WOAKES, M.D. LOND.

Senior Aural Surgeon, London Hospital; Lecturer on Diseases of the Ear, London Hospital Medical College.

ON DEAFNESS, GIDDINESS AND NOISES IN THE HEAD. Fourth Edition, with Illustrations, 8vo.

[Now ready.]

HENRY WOODS, B.A., F.G.S.

[See Cambridge Natural Science Manuals, page 5.]

HERMANN VON ZEISSL, M.D.

Late Professor at the Imperial Royal University of Vienna.

OUTLINES OF THE PATHOLOGY AND TREATMENT OF SYPHILIS AND ALLIED VENEREAL DISEASES. Second Edition, revised by M. VON ZEISSL, M.D., Privat-Dozent for Diseases of the Skin and Syphilis at the Imperial Royal University of Vienna. Translated, with Notes, by H. RAPHAEL, M.D., Attending Physician for Diseases of Genito-Urinary Organs and Syphilis, Bellevue Hospital, Out-Patient Department. Large 8vo, 18s.

OSWALD ZIEMSEN, M.D.

Knight of the Iron Cross, and of the Prussian Order of the Crown.

THE TREATMENT OF CONSTITUTIONAL SYPHILIS. Post 8vo, 3s. 6d.

Lewis's Diet Charts.

Price 5s. per packet of 100 charts, post free.

A suggestive set of diet tables for the use of Physicians, for handing to patients after consultation, modified to suit individual requirements, for Albuminuria, Anæmia and Debility, Constipation, Diabetes, Diarrhœa, Dyspepsia, Eczema, Fevers, Gall Stones, Gout and Gravel, Heart Disease (chronic), Nervous Diseases, Obesity, Phthisis, Rheumatism (chronic), and Blank Chart for other diseases.

A special leaflet on the Diet and Treatment of Infants is sold separately, price 7s. 6d. per 100, or 1s. per dozen, post free.

Lewis's Four-Hour Temperature Charts.

25s. per 1000, 14s. per 500, 3s. 6d. per 100, 2s. per 50, 1s. per 20, carriage free.

This form has been drawn up to meet the requirements of a chart on which the temperature and other observations can be recorded at intervals of four hours. They will be found most convenient in hospital and private practice. Each chart will last a week.

Clinical Charts for Temperature Observations, etc.

Arranged by W. RIGDEN, M.R.C.S. 50s. per 1000, 28s. per 500, 15s. per 250, 7s. per 100, or 1s. per dozen.

Each Chart is arranged for four weeks, and is ruled at the back for making notes of Cases; they are convenient in size, and are suitable both for hospital and private practice.

Lewis's Clinical Chart, specially designed for use with the

Visiting List. This Temperature Chart is arranged for four weeks and measures 6 × 3 inches. 30s. per 1000, 16s. 6d. per 500, 3s. 6d. per 100, 1s. per 25, 6d. per 12.

Lewis's Nursing Chart.

25s. per 1000, 14s. per 500, 3s. 6d. per 100, 2s. per 50, or 1s. per 20.

These Charts afford a ready method of recording the progress of the case from day to day. Printed on both sides.

Boards to hold the Charts, price 1s.

Chart for Recording the Examination of Urine.

40s. per 1000; 25s. per 500; 15s. per 250; 7s. 6d. per 100; 1s. per 10.

These Charts are designed for the use of Medical Men, Analysts, and others making examinations of the urine of patients, and afford a very ready and convenient method of recording the results of the examination.

LEWIS'S PRACTICAL SERIES.

Under this title a Series of Monographs is published, embracing the various branches of Medicine and Surgery. The volumes are written by well-known Hospital Physicians and Surgeons, recognized as authorities in the subjects of which they treat. The works are of a THOROUGHLY PRACTICAL nature, calculated to meet the requirements of the practitioner and student, and to present the most recent information in a compact form.

THE TREATMENT OF PULMONARY CONSUMPTION.

By VINCENT D. HARRIS, M.D. LOND., F.R.C.P., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, etc., and E. CLIFFORD BEALE, M.A., M.B. CANTAB., F.R.C.P., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, etc. Crown 8vo. 10s. 6d. [Now ready.]

THE SURGICAL DISEASES OF CHILDREN AND THEIR TREATMENT

BY MODERN METHODS. By D'ARCY POWER, M.A., M.B. Oxon., F.R.C.S. Eng., Demonstrator of Operative Surgery at St. Bartholomew's Hospital, etc. With Illustrations, crown 8vo, 10s. 6d. [Now ready.]

DISEASES OF THE NOSE AND THROAT.

By F. de HAVILLAND HALL, M.D., F.R.C.P. Lond., Physician to Out-patients, and in charge of the Throat Department at the Westminster Hospital. With coloured plates and wood engravings, crown 8vo. 10s. 6d.

PUBLIC HEALTH LABORATORY WORK.

By HENRY R. KENWOOD, M.B., D.P.H., F.C.S., Instructor in the Hygienic Laboratory, University College, &c. Second Edition, with Illustrations, cr. 8vo, 10s. 6d. [Now ready.]

MEDICAL MICROSCOPY: A GUIDE TO THE USE OF THE MICROSCOPE IN MEDICAL PRACTICE. By FRANK J. WETHERED, M.D., M.R.C.P., Demonstrator of Practical Medicine in the Middlesex Hospital Medical School, &c. With Illustrations, crown 8vo, 9s.

MEDICAL ELECTRICITY. A PRACTICAL HANDBOOK FOR STUDENTS AND PRACTITIONERS. By H. LEWIS JONES, M.A., M.D., F.R.C.P. Second edition, with Illustrations, crown 8vo, 10s. 6d.

HYGIENE AND PUBLIC HEALTH.

By LOUIS C. PARKES, M.D., D.P.H. LOND. UNIV., Fellow of the Sanitary Institute, and Member of the Board of Examiners. Fourth edition, with numerous Illustrations, cr. 8vo, 10s. 6d.

MANUAL OF OPHTHALMIC PRACTICE.

By C. HIGGINS, F.R.C.S., Ophthalmic Surgeon to Guy's Hospital; Lecturer on Ophthalmology at Guy's Hospital Medical School. Illustrations, cr. 8vo, 6s.

A PRACTICAL TEXTBOOK OF THE DISEASES OF WOMEN.

By A. H. N. LEWERS, M.D. Lond., M.R.C.P., Obstetric Physician to the London Hospital, etc. Fourth Edition, with Illustrations, crown 8vo, 10s. 6d.

ANÆSTHETICS THEIR USES AND ADMINISTRATION.

By DUDLEY W. BUXTON, M.D., B.S., M.R.C.P., Administrator of Anæsthetics and Lecturer in University College Hospital, etc. Second Edition, with Illustrations, crown 8vo, 5s.

ON FEVERS: THEIR HISTORY, ETIOLOGY, DIAGNOSIS, PROGNOSIS, AND TREATMENT. By ALEXANDER COLLIE, M.D. Aberd., M.R.C.P. Illustrated with Coloured Plates, crown 8vo, 8s. 6d.

HANDBOOK OF DISEASES OF THE EAR FOR THE USE OF STUDENTS AND PRACTITIONERS. By URBAN PRITCHARD, M.D. Edin., F.R.C.S. Eng., Professor of Aural Surgery at King's College, London. Third Edition, with Illustrations, crown 8vo, 6s. [Now ready.]

A PRACTICAL TREATISE ON DISEASES OF THE KIDNEYS AND URINARY DERANGEMENTS. By C. H. RALFE, M.A., M.D., F.R.C.P., Assistant Physician to the London Hospital, etc. Illustrations, cr. 8vo, 10s. 6d.

DENTAL SURGERY FOR MEDICAL PRACTITIONERS AND STUDENTS OF MEDICINE. By ASHLEY W. BARRETT, M.B. Lond., M.R.C.S., L.D.S., Dental Surgeon to, and Lecturer on Dental Surgery in the Medical School of, the London Hospital. Third edition, with Illustrations, cr. 8vo, 3s. 6d. [Now ready.]

BODILY DEFORMITIES AND THEIR TREATMENT: A HANDBOOK OF PRACTICAL ORTHOPÆDICS. By H. A. REEVES, F.R.C.S. Edin., Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital, etc. With numerous Illustrations, cr. 8vo, 8s. 6d.

THE NEW SYDENHAM SOCIETY'S PUBLICATIONS.

President :—HERMANN WEBER, M.D.

Honorary Secretary :—JONATHAN HUTCHINSON, ESQ., F.R.S.

Treasurer :—W. SEDGWICK SAUNDERS, M.D., F.S.A.

Annual Subscription, One Guinea.

The Society issues translations of recent standard works by continental authors on subjects of general interest to the profession.

Amongst works recently issued are "Binz's Pharmacology," "Sir William Gull's Collected Works," "Laveran's Paludism," "Pozzi's Gynecology," "Flügge's Micro-Organisms," "Cohnheim's Pathology," "Henoch's Children," "Spiegelberg's Midwifery," "Hirsch's Historical and Geographical Pathology," "Ewald's Disorders of Digestion," works by Charcot, Duchenne, Begbie, Billroth, Graves, Koch, Hebra, Guttman, etc.

The Society also has in hand an Atlas of Pathology with Coloured Plates, and a valuable and exhaustive "Lexicon of Medicine and the Allied Sciences."

The Annual Report, with full list of works published, and all further information will be sent on application.

PERIODICAL WORKS PUBLISHED BY H. K. LEWIS.

THE BRITISH JOURNAL OF DERMATOLOGY. Edited by William Anderson, H. G. Brooke, H. Radcliffe Crocker, T. Colcott Fox, Stephen Mackenzie, Malcolm Morris, J. F. Payne, J. J. Pringle and James Galloway. Published monthly, 1s. Annual Subscription, 12s. post free.

THE BIRMINGHAM MEDICAL REVIEW. Edited by J. W. Russell, M.D., and and O. J. Kauffmann, M.D. Published monthly, 6d. Annual Subscription, 6s. post free.

TRANSACTIONS OF THE DERMATOLOGICAL SOCIETY OF GREAT BRITAIN AND IRELAND. Vols. I. and II., royal 8vo, 5s. each.

THE THERAPEUTIC GAZETTE. A Monthly Journal, devoted to the Science of Pharmacology, and to the introduction of New Therapeutic Agents. Edited by Dr. R. M. Smith. Annual Subscription, 10s., post free.

THE GLASGOW MEDICAL JOURNAL. Published Monthly. Annual Subscription, 20s., post free. Single numbers, 2s. each.

LIVERPOOL MEDICO-CHIRURGICAL JOURNAL, including the Proceedings of the Liverpool Medical Institution. Published twice yearly, 3s. 6d. each number.

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA. Volumes I. to VI., 8vo, 10s. 6d. each.

MIDDLESEX HOSPITAL, REPORTS OF THE MEDICAL, SURGICAL, AND Pathological Registrars for 1883 to 1894. Demy 8vo, 2s. 6d. *nett* each volume.

ARCHIVES OF PEDIATRICS. A Monthly Journal, devoted to the Diseases of Infants and Children. Edited by Dr. W. P. Watson. Annual Subscription, 12s. 6d., post free.

* * MR. LEWIS is in constant communication with the leading publishing firms in America, and has transactions with them for the sale of his publications in that country. Advantageous arrangements are made in the interests of Authors for the publishing of their works in the United States.

Mr. Lewis's publications can be procured of all Booksellers in any part of the world.

